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EDITORIALS

KEEPING SURGEONS OUT OF COURT

SURGEONS find it extremely difficult to take vacations. Yet they are often forced to take time away from their patients and wait for hours, even days, to give legal testimony that ordinarily consumes but five or ten minutes. Aside from the possible danger to the lives of their patients through their absence, their practices suffer severely. Furthermore, any compensation which they may receive is hardly adequate for the loss sustained.

Surgeons might well organize to attempt to promote legislation similar to that which now exists in the federal courts, in order to keep them from the witness chair. In New York, a physician's certificate, properly sworn to, is accepted as evidence in compensation cases. It should be so in all cases—and in all states.

Physicians should be permitted to give their testimony at their convenience, in their own offices, before a notary public or commissioner of deeds, instead of having to make personal appearances in court. Attorneys should be permitted to cross examine at such examination. The procedure, in most cases, would not consume more than fifteen minutes and would not conflict with the surgeon's practice.

The profession could seek an amendment to the rules of evidence that would permit them to give their testimony on the foregoing basis. Surgeons should not be forced into court as professional witnesses in a controversy in which they have no personal interest, except to tell what they may know. No surgeon should be compelled to forsake the operating table for the counsel table.

Meyer Kirschenbaum, Member of the New York Bar.

ALLEN DEMING LAZENBY

1894–1939

IT is with regret that we announce the death on April 18th of Allen Deming Lazenby. He was 45 years old, had already behind him a notable career, and gave promise of even greater achievements.

Dr. Lazenby, a graduate of the University of Maryland School of Medicine and College of Physicians and Surgeons, was an outstanding worker in the industrial health field. He was one of the original members of the Council on Industrial Health of the American Medical Association, a Fellow of the American College of Surgeons and of the Association of Industrial Physicians and Surgeons. In addition, he was chairman of the Industrial Health Committee of the Medical and Chirurgical Faculty of Maryland; a member of the Medical Committee of the New York World's Fair, and vice-president and medical director of the Maryland Casualty Company. He had been a captain in the Medical Corps of the United States Army, but had retired from active duty in 1919 for disability incident to the service.

Dr. Lazenby acted as Guest Editor of the Traumatic and Industrial Surgery Symposium published as the December, 1938 issue of The American Journal of Surgery. As a result of his efforts, his knowledge of the subject and his familiarity with eminent authorities in this field, this Symposium has become a standard text for physicians throughout the country and a work of reference invaluable to many lay agencies connected with the field of compensation, industrial and traumatic medicine.

Too young to go, yet during his short life Allen Lazenby had left the impression of his ability and personality in the sphere of his professional activity.

To his family, the Publisher and Editor of The American Journal of Surgery offer their deepest sympathy. T. S. W.

CORRECTION

Dr. Arthur Steindler wishes us to call attention to an inadvertent error in the labeling of Figure 3B of his article on "Tendon Transplantation in the Upper Extremity," page 268 of the April, 1939 issue. "Extensor carpi ulnaris" should have been "flexor carpi ulnaris"; "extensor carpi radialis" should have been "palmaris longus"; and "palmaris longus" should have been "flexor carpi radialis."

ORIGINAL ARTICLES

THE SURGICAL TREATMENT OF ESSENTIAL HYPERTENSION

PAUL G. FLOTHOW, M.D.

SEATTLE, WASHINGTON

THE etiology of essential hypertension is unknown. Without doubt there are many factors involved in its production. The endocrine glands and the sympathetic nervous system are certainly related in a definite manner in its production. It is well known that the thyroid, pituitary and adrenal glands all play an important part in production of vasomotor and vasopressor phenomena. It is probable that there are definite cerebral centers, perhaps in the diencephalon, which exert regulatory effects on blood pressure levels. Heredity and environment must play a further important part in the picture.

The importance of the study, understanding and hopeful therapy of this condition are little appreciated. By virtue of the fact, perhaps, that it has been so long considered a hopeless condition and one for which so little can be done, we have adopted a defeatist attitude toward the problem. Statistics of the Metropolitan Life Insurance Company show that four times as many people die annually in the United States from conditions in which hypertension is either the cause or a contributing factor that die from cancer. We have cancer clinics and associations for the study of and prevention of cancer, lay organizations for the control and early recognition of cancer, yet nothing similar is being done in the case of hypertension.

While the time has been too short to give authoritative statistics, various reports show that following surgical removal of various visceral nerves, from 25 to 50 per cent of patients with essential hypertension have had blood pressure levels returned approximately to normal. This certainly compares very favorably with the results in the treatment of cancer.

There are four schools of thought regarding the surgical attack upon essential hypertension, namely, those of the Mayo Clinic, Peet, Crile and Heuer.

In general it may be said that all of the men engaged in this type of work agree that it is far from being a satisfactory answer to the problem of treatment of this condition. The concensus of opinion is, that while it cannot be considered a cure, it is at this time by far the most effective method of treatment, and the work should continue until some better form of therapy is found.

The viewpoints of the surgeon and the internist differ somewhat as to the value of this method of treatment. Internists who have seen only the occasional, and frequently the unsuccessful, case are very free with their criticism of the procedure. Surgeons who have done a great deal of this work, while not entirely enthusiastic about the results, are unanimous in feeling that their results render the continuation of this work well worth while. Strangely enough, some of the most enthusiastic supporters are internists in institutions where work of this kind is being carried on: men who have had opportunity of seeing the patients before and after surgery and of following the postoperative results.

The reviewer has recently visited the Mayo Clinic, the Cleveland Clinic and the

University Hospital at Ann Arbor, three centers where a great deal of the work in this country has been done. He has interviewed both the internists and the surgeons, has inspected the case reports, the follow-ups and the statistical reports of the results of the various types of surgery. It has been an amazing observation that regardless of what the surgical approach has been, and almost regardless of the manner of selection of cases, the results have been very similar in these three different institutions.

The manner of selection of cases varies at each institution. In one a great deal of attention will be paid to a particular point as a criterion of suitability for operation, while in another it will be an entirely different one.

The results are certainly good enough to warrant the continued application of visceral nerve surgery in selected cases of hypertension.

METHOD OF SELECTION

Cases are very carefully selected. They are usually selected by the internist and not by the surgeon. The preoperative studies are made on the medical side, and the internist determines whether or not the neurosurgeon should be offered the case. Very careful preoperative studies are carried out. These include the following:

1. Careful examination of the heart including electrocardiographic studies, *x*-rays and clinical examination.
2. Careful tests of kidney function, including all of the well-known tests, particularly the concentration test.
3. Careful determination of blood pressure levels with hourly determinations for a period of at least forty-eight hours.
4. The determination of the effect of large doses of sodium amytal or nembutal upon the blood pressure levels.
5. The effect of sodium nitrite on the blood pressure levels.
6. The cold test.
7. The intravenous pentothal test.

The age of the patient, the condition of the blood vessels and the duration of symptoms and rapidity of progress are also taken into consideration.

Marked cardiac damage is a contraindication to surgery. However, many patients with essential hypertension show a certain amount of cardiac damage, and it becomes the task of the internist to determine whether this damage is irreparable. Some patients in whom serious heart complications are present have shown tremendous improvement after operation. The estimation of whether or not cardiac function will improve is a very difficult one, and rather severe cardiac damage should not absolutely contraindicate the operation, if other factors are favorable.

Severe kidney damage with marked reduction of renal function serves in most cases to contraindicate operation. However, the various tests of renal function do not tell the entire story. Peet believes the concentration test is of great importance and feels that if the kidneys are unable to concentrate the urine, operation offers very little hope. When other tests of kidney function such as the various protein tests and the P. S. P. test show marked reduction of function, but the kidney is still able to concentrate the urine, he feels that the kidneys are better than is apparent from the other tests. The presence of albumin apparently is not of great significance in regard to actual kidney function.

Allen and Adson[2] make the following statements: "We do not consider apparent sclerosis of the retinal arteries, moderate enlargement of the heart, inversion of the T-waves in the electrocardiograms, albuminuria, slight reduction in renal function or cerebral vascular accident from which recovery has been satisfactory, contraindications, in themselves, to operation. However, we do not advise operation for patients who have congestive heart failure, marked renal insufficiency, advanced arteriosclerosis, or angina pectoris."

While the determination of preoperative blood pressure levels is carried out in all institutions, some pay much more attention to this factor than others.

The effect of sodium amytal, 9 to 12 gr. (0.58 to 0.77 Gm.) or more, or nembutal in sufficient quantities to insure the patient's sleeping through the taking of blood pressures, is considered of considerable importance. Suitable cases should show a marked drop in the resting blood pressure levels, and it has been the reviewer's experience that the final postoperative blood pressure levels very closely approximate the resting figures.

Sodium nitrite given in ½ gr. (0.03 Gm.) doses, repeated every half hour for three hours, should have an appreciable effect in lowering the blood pressure to a basal level.

The cold test is performed by having the patient immerse the hand up to the wrist in ice water to which salt has been added to bring the temperature to 4°c. Several blood pressure readings are taken immediately before placing the hand in the ice water to determine the mean pressure. One hand is then immersed and the blood pressures taken every half minute for from two to ten minutes. The hand should be kept in the water for one minute if possible, but some patients cannot bear it more than thirty seconds. A response of increase in blood pressure of not over ten points is considered normal; from ten to twenty points slightly abnormal, and anything above this a markedly abnormal reaction. In some cases the systolic blood pressure may rise as much as fifty points as a result of the immersion of the hand in cold water. It is felt that this reactive rise in blood pressure indicates the relative importance of vasoconstriction as a factor in the individual case, and the higher the rise in the systolic pressure, the more favorable is it considered as an index of the suitability for operation.

The intravenous pentothal sodium test may be considered as the reverse of the cold test. It is felt that where nerve impulses play an important part in the production of elevated blood pressures, abolition of these impulses by an intravenous anesthetic such as pentothal sodium should result in a marked fall in the blood pressure levels. It would appear, therefore, that a patient who demonstrates a marked fall would be a suitable one, whereas one in which the fall was slight should most certainly be unsuitable.

The test is simply performed, it being merely a matter of establishing the patient's blood pressure in a fixed position, preferably sitting or lying in bed, and then slowly administering the solution until the patient has completely lost consciousness. The blood pressure levels are determined at frequent intervals during this process until the patient regains consciousness, or until the blood pressure returns to the original level. It is felt that perhaps the blood pressure at the depth of complete unconsciousness is the basic level for that individual, and it could not be hoped that an operation would produce a lower sustained level.

Allen and Adson[2] state that incomplete studies indicate that pressure on the carotid sinuses may affect blood pressure to much the same degree that rest, amytal, sodium nitrite and pentothal affect it.

The age of the patient undoubtedly is of great importance, and, in general, the younger the patient, the better the possibilities. Some institutions established 40 as a maximum age earlier in their work, but now they have raised it to approximately 50. Crile does not feel that age is important, or rather, he feels that age does not necessarily contraindicate operation. He regards the degree of arteriosclerosis as much more important than the age of the individual. Most authorities believe that 50 years is just about the maximum age, and that patients over that age offer very little chance of improvement.

Any marked degree of arteriosclerosis is universally felt to be a definite contraindication to surgery.

There seems to be no uniformity of opinion regarding the importance of duration of symptoms and rapidity of progress. In general, it may be stated that most authorities are of the opinion that cases in which hypertension has been present a relatively short time and has progressed rather rapidly, are less favorable than those

of many years' standing with a relatively slow rise of pressure levels. Those of very short duration with a very rapid rise in blood pressure levels usually are in the very malignant stage when first seen, and are, therefore, unsuitable.

The presence or absence and the degree of changes in the retinal arteries and optic discs are considered very important. Early retinal changes and minor degrees of spasm of retinal vessels can be determined only by skilled ophthalmologists versed in this particular field. Retinal changes severe enough to be noted by the occasional observer, frank choking of the optic discs, or retinal hemorrhage, are all evidences of malignancy, and indicate that the chance of surgery being of value is rather remote.

The reviewer believes that the entire future of this field of surgery is dependent upon the proper selection of cases. It is absolutely necessary that definite criteria of selection be established by means of which we may accurately select cases and foretell the approximate results.

The major purpose of the visits to the before-mentioned Clinics was to determine whether or not such criteria had been, or could be established. It is unfortunate, indeed, that one must report that at the present time this cannot be done. There are two broad methods of error. First of all, even though all of the tests may indicate that a case is highly suitable for surgery, yet the best we can hope for is just a little better than a 50 per cent chance of a good result. On the other hand, occasionally a case that would seem to be absolutely hopeless from the standpoint of these tests when operated turns out to have an excellent result. Therefore, it becomes impossible to determine accurately either one way or the other whether a case is suitable or unsuitable, or what our final result will be.

This much may be said, however. The chance of a good result in a patient who responds poorly to all or most of the tests is minimal. Operations in this type of case should rarely be undertaken. If we could be as accurate in determining which cases should be done as in determining which should not be done, our percentage of good results would be greatly increased.

The reviewer has made certain observations and has come to noteworthy conclusions regarding the selection of cases for operation. It has been our experience in a limited number of cases, just as it has been the experience of all others engaged in this line of work, that the "batting average" in any one test as compared with the final result is not much better than 50 per cent. We feel, however, that the determination of resting blood pressure levels offers a very accurate index. The patient whose blood pressure level does not fall at night when determinations are made without disturbing the patient, in our estimation offers a very poor subject for a good result following surgery regardless of what the result may have been of any of the other tests. We feel that this affords an accurate index of the effect of nervous and vasomotor impulses on the blood pressure.

The cold test may produce a marked rise, but does not indicate what may be expected in regard to a fall in blood pressure.

The intravenous pentothal test introduces a profound chemical reaction, the effect of which may be such upon the central nervous system as cannot be approximated by surgery.

It has been our observation that those cases that did not show a fall in resting blood pressure levels have shown no appreciable fall postoperatively, and we, therefore, hesitate very much to advise operation in cases of this type regardless of what the other tests may show.

CAUSES OF HYPERTENSION

It is well to call attention to the fact that there are conditions other than essential hypertension which may produce high blood pressure. The following classification is taken from Adson and Allen:[2]

1. Primary or essential hypertension. Estimated 85 per cent of all cases.
2. Secondary hypertension due to known disease. Estimated 15 per cent of all cases.

(*a*) Coarctation of the aorta.
(*b*) Glomerular nephritis.
(*c*) Tumors of the suprarenal glands.
(*d*) Hyperthyroidism.
(*e*) Arteriosclerosis.
(*f*) Traumatic arteriovenous fistula.
(*g*) Aortic heart disease.

Essential hypertension may be roughly divided into three classes:

1. Chronic, which is slowly progressive, many years elapsing before it becomes malignant.

2. Subacute in which there is a comparatively short history and only a few years elapse from onset to malignant changes.

3. Acute in which the condition is rapidly progressive and probably is either malignant from onset or rapidly becomes so.

Blood pressure levels are not necessarily the criterion of malignancy, but kidney, cardiac and retinal damage are so considered. Some cases in Group 1 do not need surgery. Many of the cases in Group 2 are suitable for surgery, but only a few in Group 3.

The entire question of operating for hypertension can be likened to that of operation for cancer of the stomach, for example. Most cases of cancer of the stomach are inoperable when discovered as far as a reasonable chance of cure is concerned. No one would think of criticizing a surgeon for exploring a cancer of the stomach if there was any hope of cure or even palliation, yet these same individuals will throw up their hands in "holy horror" when an operation for hypertension is proposed, notwithstanding the fact that the results are not even comparable either from the standpoint of cure or palliation, and hypertension is just as deadly as cancer of the stomach.

Since we adopt heroic measures in one fatal condition, why condemn heroic measures in another where the chances of benefiting the patient are infinitely better?

SURGICAL TECHNIQUES

Supradiaphragmatic Approach. In this operation the splanchnic and sympathetic nerves are resected above the diaphragm. The question of whether or not this is the proper or a superior approach depends upon the determination of whether or not fibers from the first and second lumbar sympathetic ganglia play any part in the product of elevated pressures. Peet[4] states that they do not. The Mayo Clinic group[1,2] feel that they play an important part in the innervation of the blood vessels of the kidney and it is, therefore, necessary that they be removed.

As far as the nerve supply to the celiac ganglion and plexus is concerned, Peet's operation is entirely preganglionic, and, if the same thing is true in this instance as has apparently been proved to be the case in Raynaud's disease, this approach theoretically should be the most favorable. It has a further advantage in that both sides can be done at one operation. Its theoretical disadvantages are the inability to denervate the first and second lumbar ganglia and the fact that the fibers of the aortic plexus coming through the diaphragm with the aorta, which may play a considerable part in the production of vasospastic phenomena, are not interrupted. It has the further disadvantage that the adrenal glands may not be explored and the occasional case of tumor of the adrenal glands may be overlooked.

It has not been determined whether or not a preganglionic operation is superior to a postganglionic operation in the case of hypertension. The final determination must come after a period of years and a careful comparison of results.

Adson-Craig Type of Operation.[1] The technique of this operation has been described frequently. It consists of a subdiaphragmatic approach with or without the removal of a portion of the twelfth rib, the removal of the major, minor and least splanchnic nerves, and a portion of the celiac ganglion. In addition to this the first and second lumbar ganglia are removed and the dissection carried up into the diaphragm from below. Recently, they have at times been removing the entire celiac ganglion. Previously, a portion of the

adrenal gland was removed, but they have abandoned this procedure, being content to explore the adrenal glands to determine the presence or absence of a tumor.

The disadvantages of this operation are that it must be done at two stages, and that it is a considerably more difficult surgical procedure than that of Peet's. This operation is somewhat a hybrid, part of it being pre- and part of it being post-ganglionic. If the entire celiac ganglion is removed, it becomes a truly postganglionic operation, and as before stated this offers a theoretic disadvantage. It is apparently definitely safer than the Peet operation, a large number of these cases having been done at the Mayo Clinic without any deaths, whereas in Peets series there has been a mortality of approximately 4 per cent.

Crile Operation. This may be considered a modification of the Adson-Craig operation. A small incision just large enough to admit the hand is made in the flank just below the twelfth rib, carried forward and downward into the retroperitoneal space. He identifies the celiac ganglion by sense of touch, places a hook on it and removes the ganglion by cutting against his finger with a long scissors, and at no time visualizes the structure involved. In addition to this he states that he denervates the adrenal gland with his finger. The lumbar sympathetics are not disturbed, and the various structures are not individually identified.

The advantage of this operation is the rapidity with which it may be done. Its disadvantage is that it may very readily be incomplete as far as denervation of the celiac plexus is concerned. In Crile's hands it is apparently an excellent operation. It is doubtful, however, that other surgeons could perform it with equal success. He has done a large series of cases, approaching one hundred, without a death, although previous to that series he had several deaths.

The Intradural Section of the Anterior Root Components of the Splanchnic Nerves (*Heuer*[5]). This operation requires a section of all the anterior roots from the fifth dorsal to the second lumbar, thus necessitating a very long laminectomy, and while the results in some of these cases have been excellent, the magnitude of the surgical procedure offers its greatest disadvantage.

The reviewer has performed a modification of the Adson-Craig operation starting with his first case, namely, the removal of the entire celiac ganglion, and recently section of the aortic plexus medial to the ganglion. This is entirely a postganglionic operation and the reviewer feels that it accomplishes complete denervation of all vasomotor fibers on the postganglionic side. In all other respects the operation is exactly the same as the Adson-Craig operation, the rib being rarely removed. There have been no surgical deaths.

RESULTS OF SURGERY

It is quite amazing that regardless of the type of operation performed, all of the groups report approximately the same results, namely, about 50 per cent good results and 50 per cent poor. Among those classed as poor, there are approximately half who receive clinical and symptomatic improvement even though their blood pressures are not affected.

The Mayo Clinic reports 25 per cent excellent results, 30 per cent good results, and 45 per cent poor results. Of the 45 per cent poor results approximately one-half show symptomatic improvement.

Peet's[4] results are very similar to those of the Mayo Clinic. The two institutions have about the same number of cases, and the results measured from all standpoints are almost parallel.

Crile's results are rather difficult to evaluate since he uses the decrease in diastolic pressure as his criterion of results and disregards the systolic pressure, while other clinics use the drop in systolic pressure as their basis. On the whole, however, his percentage of success is about the same as the preceding.

In a recent report from the Lakeside Hospital[7] a series of twelve cases were

reported in which Heuer's operation had been done, most of them with poor results.

RESULTS

A statistical analysis of our own results is of necessity rather difficult by virtue of the type of cases submitted to operation. Twenty-two are included, the others being too recent to evaluate.

When we first started this procedure we operated on patients whom we would not consider as subjects now. In fact, in the past year more cases have been refused than accepted. Of the twenty-two, eight were in the definitely malignant stage, and at present at least five of these would have been refused. Two of these five have had results which justified surgery. Two others would have been refused because of their age. One has had a good result, and the other has since succumbed.

The cases will be considered first in two groups, and then as a whole to demonstrate our percentage of success. In the first group are ten cases which, due either to malignancy of disease or the age of the patient, would be questionable cases, and at least five of them would not be considered for surgery at the present time. Four of these ten have since died of their disease, and one of postoperative pneumonia following a hysterectomy. There have been no deaths attributable to the operation in the entire series. Two of the five survivors in this group have exhibited marked reduction of their blood pressure levels, two have been remarkably improved clinically without marked drop in pressure, and one has had no improvement either in clinical symptoms or in pressure levels. Two cases, or 20 per cent of the ten in this group have had marked reduction in blood pressure levels, and two cases, or 20 per cent, have been markedly improved clinically which makes a total of 40 per cent in which surgery has proved beneficial.

One of the cases in this group is particularly noteworthy. The patient was a woman 51 years old with a fifteen year history of blood pressure above 200 which was gradually mounting. When first seen she had been bedridden for five months because of severe headache and palpitation on the slightest exertion. Her resting pressure was 300 plus systolic, and 156 diastolic. There was definite cardiac and kidney involvement, and she was in the last stages of her disease. Intravenous pentothal sodium dropped the systolic pressure from 285 to 185. Both patient and family requested operation in spite of the poor prognosis. She made an excellent recovery, and within three months was doing her own housework, and was practically free of symptoms except when she overexerted herself. She continued in excellent health for over two years with her blood pressure ranging between 190 and 225 systolic. Within the past month, after a long period of overwork, she has again flared up with a pressure of 285, and return of many of her previous symptoms, although she is still clinically considerably better than she was before the operations. This patient would undoubtedly have expired long ago had she not been operated upon.

Of the twelve patients in the second or definitely favorable group both as to age and diagnostic results of the various tests, there are six who have exhibited marked sustained falls in pressure, and six who have shown little or no fall in blood pressure, but have had marked clinical improvement. Fifty per cent of these cases are, therefore, classed as very successful, and 50 per cent as moderately successful. In the entire group the result has been satisfactory, and the operations have proved well worthwhile in all the cases as far as the patient is concerned.

Eight patients in the entire series of twenty-two, or 36.3 per cent, have shown marked sustained falls in blood pressure levels. Eight, or 36.3 per cent, have shown little fall in blood pressure levels, but a marked clinical improvement. The remaining six cases, or 27.4 per cent have been classed as failures. Five of these six cases were in the unfavorable group which would

not be accepted for operation at the present time.

Too frequently opinions as to the value of this treatment are based upon the observation of those cases which might be classed as failures by reason of the fact that their blood pressures are not materially lowered. Those of us doing this work, however, cannot be convinced that it is not of value since we have all had experience with cases in which the results have been excellent.

In all of the author's cases it has been almost invariably true that the patients have been clinically and symptomatically markedly improved regardless of the effect on blood pressure readings. We have come to the conclusion that the results of the operation cannot be measured by manometric blood pressure determinations, and that many cases, which for purposes of statistics are classified as fair or poor results, should not be so classified. We feel that the operation produces results upon factors in the underlying causes and the associated effects of hypertension which we have no means of measuring.

One case may be cited as an example of such effects. A man, age 32, who had been discharged from the Navy because of hypertension and the fact that he was suffering from severe headaches, shortness of breath, fatigability, etc., desired operation. All of the preoperative diagnostic tests indicated that his should be an unfavorable result, and he was advised against the operation. He insisted upon it, however, and offered himself as an experimental case. Before he left the hospital his blood pressure was 260/160 which was exactly the same as his preoperative levels. Four months later this man came in stating that he felt as well as he ever had in his life, and asked for a letter to the Navy recommending reinstatement. He said that he could do as good a day's work as he had ever done, and that he had no complaints or symptoms. His blood pressure was still 260/160. His eye grounds, which had showed hemorrhages before, were entirely clear, and in every way except for his blood pressure readings this man was perfectly well.

One thing seems quite certain; namely, that even though postoperative pressure may approximate preoperative pressures, the extreme pinnacles of pressure that had occurred before have been eradicated, as well as many or all of the subjective symptoms of the disease.

Since there is no other type of treatment which offers results in any way comparable to that of the surgical treatment, the work should certainly be continued, and perhaps as time goes on, definite criteria may be evolved for more accurate selection of cases so that failures will become less and less frequent.

SUMMARY

The various tests made to select suitable cases of essential hypertension for surgical treatment are described and their value discussed.

An attempt is made to indicate the proper cases for operation, and the indications and contraindications set forth.

The various operative techniques are described and their advantages and disadvantages discussed.

The results of the various types of surgery and of the groups doing this work are reported. Individual groups report similar results. Excellent results are reported in 25 to 50 per cent, and worthwhile results in over 50 per cent of cases by all groups. In addition all reports show a marked improvement clinically without fall in blood pressure in approximately another 25 per cent of cases.

The author reports 36.3 per cent excellent results, 36.3 per cent good results and 27.4 per cent failures in a series of twenty-two cases.

CONCLUSIONS

The surgical treatment of hypertension has now been practiced long enough to

prove that it is of great value in a selected group of cases. When cases are carefully selected, surgery offers at least an even chance of proving markedly beneficial.

As time goes on and criteria of selection become more accurate, the percentage of successful results should be markedly increased.

There is no other method of treatment of essential hypertension that offers anything even comparable to the results following extensive sympathectomy. Without doubt life can be prolonged and many patients markedly benefited by surgical procedures in essential hypertension.

Postoperative blood pressure cannot be the only criterion of results accomplished since many patients who exhibit no fall in blood pressure are markedly improved clinically and symptomatically.

The various objections that have been made to the operative treatment of essential hypertension are well answered by Allen and Adson.[6]

Only time will answer the question of whether or not the operations result in permanent cure, but there is no doubt as to their value in many cases of several years' duration.

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HYDATIDIFORM MOLE*

WITH ANALYSIS AND FOLLOW-UP OF TWENTY-FOUR CASES

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A TYPICAL hydatidiform mole consists of a mass of translucent, grape-like vesicles, arranged in stalks, similar to beads on a string. The vesicles contain a clear, viscid substance and vary in size from the microscopic to about 3 cm. in diameter. They result from the degeneration of chorionic villi, so that each stalk represents what was once a villus. This peculiar formation usually occurs early in pregnancy when the ovum is surrounded by chorionic villi. Ordinarily it involves the entire periphery of the chorionic membrane but at times is limited to portions of it.

HISTORY AND PATHOLOGY

It is generally stated that the condition was first described by von Grafenberg in 1565, but it has since been pointed out that Aetius of Amida, in the early part of the sixth century, wrote intelligently about hydatidiform mole, although he seemed to have no clear idea as to its true nature.

As the name implies, many of the early writers considered the condition analogous to the echinococcus or hydatid cysts observed in other parts of the body. Some held that the vesicles were mature ova; others that they were multiple embryos.[1] It was not until the nineteenth century that the true nature of the lesion was finally recognized.

In 1895, Marchand[2] demonstrated that the essential lesion was to be found not only in the stroma, but in the trophoblastic epithelial covering of the villus—the syncytial and Langhans layers. He showed that both layers undergo profuse and irregular proliferation, deeply invading Nitabuch's fibrin layer into the decidua and occasionally into and through the uterine musculature. This increase in the cellular activity of the trophoblast probably accounts for the penetration of maternal tissues, which, in normal pregnancy are not invaded.

While the trophoblast is undergoing proliferation, the blood vessels of the terminal villi are obliterated and disappear. The stroma cells degenerate and their nuclei lose their staining reaction. The stroma is replaced by a central cavity filled with fluid which has the characteristics of edema fluid rather than mucin. In consequence of this proliferation and stroma change, the resulting mass of tissue frequently reaches the size of a five months' gestation.

In his original article, Marchand[2] stated that in many instances of hydatidiform mole, the ovaries are the seat of cystic changes. These changes have also been noted by other observers. In some instances, one or both ovaries may become converted into polycystic tumors, attaining a diameter of from 10 to 15 cm. The component cysts vary from a few millimeters to 5 or 6 cm. in diameter. They are lined by lutein cells and are filled with a clear content. Although lutein cell cystomata occur fairly often in cases of hydatidiform mole, they are not a universal accompaniment of the condition. Bland[4] and Findley[3] report their occurrence in 1.5 and 11 per cent of the cases respectively, while only one of Williams' cases showed this complication.[4] Novak[4] believes that they probably are always present, but are often overlooked during routine examination, especially when very small. Most authors are of the opinion that these cystic changes represent an exaggeration of theca lutein proliferation which occurs in normal pregnancy. It is generally thought

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that they are due indirectly to the action of the proliferating trophoblast on the anterior lobe of the pituitary body. These cystoma undergo spontaneous involution within a few months after expulsion of the mole.

ETIOLOGY

The etiology of hydatidiform mole is unknown. Multiparity and endometritis may play a part in its causation.[5] It is undoubtedly a process that originates in the ovum, rather than in the maternal tissues, for in several recorded instances of twin pregnancy, one ovum was normal, while the other was the seat of a hydatidiform mole.[5]

INCIDENCE

It is generally taught that hydatidiform mole is a rare disease. According to Madame Boivin,[4] it occurs once in 20,000 cases. Later authors feel that it is of more frequent occurrence. Storch,[4] in a series of unselected, abortive ova, found that 75 per cent showed some evidence of hydatidiform change. Meyer[6] notes hydatidiform degeneration of varying degree in from 4 to 10 per cent of all pregnancies. He noted evidence of it in 42.3 per cent of all extrauterine pregnancies and in 32.4 per cent of uterine abortions. Gordon,[4] in analyzing 4,500 abortions at Bellevue Hospital, observed twenty-one cases, an incidence of 1 in 214. Velasco[4] noted its occurrence once in 204. Allen[7] states that it is the most common disease of the placenta and considers its incidence as about 1 in 300. The condition is probably much more frequent than is usually thought, because early hydatidiform changes may be overlooked by the clinician and even by the pathologist. According to Findley,[3] who presented a collective review of 500 reported cases, *clinically noticeable* hydatidiform mole occurs once in 728 pregnancies.

As to age incidence, it is essentially a disease of the active child-bearing period, that is from 20 to 35 years.[4] As to parity, it is much more common in patients who have previously borne children. A normal pregnancy following hydatidiform mole is quite common, while recurrence in subsequent pregnancies is rare,[5] it having been noted in less than 1.5 per cent of the cases.[8]

SYMPTOMS AND SIGNS

Hydatidiform mole is usually observed in the first trimester of pregnancy. Its chief symptoms are vaginal bleeding and rapid enlargement of the uterus out of proportion to the period of gestation. In some cases, abdominal pain, hyperemesis[8] or symptoms of pre-eclamptic toxemia with edema, hypertension and marked albuminuria may be present. If the growth extends through the entire wall of the uterus, symptoms of perforation and internal hemorrhage will occur.

The bleeding gives rise to a pale red or brown vaginal discharge, which often is watery in character. Profuse hemorrhage is unusual until the mole is expelled, at which time it may be alarming.

The enlargement of the uterus may be quite marked, although at times the uterus is unchanged or may even be smaller than would be expected. The latter is especially true if the patient is not examined until the second or third trimester. Thus, in a patient three months pregnant, the uterus may extend to or above the unbilicus. Owing to the absence of the fetus and liquor amnii, no ballottement is obtainable and probably for the same reason, the uterus has a peculiar, boggy consistency.

DIAGNOSIS

Fetal movements and fetal heart sounds are absent. The Aschheim-Zondek (Friedman) test is markedly positive. Vaginal examination reveals the bloody discharge and the enlarged uterus with its unusual consistency. One or both ovaries may be enlarged and cystic. Eventually, the mole is expelled spontaneously and then a few grape-like vesicles are discharged with bleeding. When vesicles are felt or seen, the diagnosis becomes certain.

PROGNOSIS

Fetal Prognosis. As a rule the embryo dies and is absorbed.

Maternal Prognosis. According to Bland,[4] one patient out of every eight or ten dies from hydatidiform mole either *directly*, as a result of hemorrhage, sepsis, perforation with peritonitis, or *indirectly*, as a result of chorionepithelioma.

Dorland[4] noted an immediate mortality of 10 per cent in a series of 100 cases which he collected from the literature. Three per cent died from hemorrhage, 2 per cent from perforation, and 5 per cent from sepsis. Findley,[3] in his analysis of 500 reported cases, gives a total mortality of 22 per cent: 4 per cent from hemorrhage; 2 per cent from perforation and sepsis, and 16 per cent from chorionepithelioma.

Figures as to the relation of chorionepithelioma to hydatidiform mole are very unsatisfactory and many writers are at variance on the subject. Bland[4] thinks that it is highly probable that malignant disease of the chorion rarely, if ever, occurs without preceding hydatidiform mole, although hydatidiform degeneration may not be recognized or even suspected. Findley[3] found that hydatidiform mole was followed by chorionepithelioma in 31.4 per cent of cases. His figures are from reported cases, and as Mathieu[23] points out, the incidence would be higher in a group of reported cases than in an unreported series. Mathieu,[23] in a recent report of 127 cases, found mole followed by chorionepithelioma in twelve patients (9.4 per cent). Schumann[10] teaches that 10 per cent are followed by chorionepithelioma. Novak[4] feels that only 1 per cent are followed by chorionepithelioma. De Lee[5] has never seen a case of mole followed by chorionepithelioma. Symmers,[5] in a follow-up of eighteen cases of hydatidiform mole, found no cases of chorionepithelioma.

TREATMENT

It is universally agreed that the uterus should be emptied as soon as a diagnosis of hydatidiform mole has been made. Delay may result in hemorrhage, perforation or sepsis.

The cervix should be dilated sufficiently to admit two fingers, and the growth peeled off the uterine wall, using great gentleness to avoid possible perforation. For the same reason, no curette or sharp instrument should be used. Following removal, the uterine cavity should be thoroughly iodinized. It is important that the growth be completely removed because retention of vesicles may lead to sepsis or continued hemorrhage. Some gynecologists prefer to empty the uterus by means of anterior vaginal hysterotomy. Schumann[10] advises abdominal hysterotomy followed by hysterectomy if necessary after examining the uterine wall. This seems to be a very radical procedure unless the patient is near the menopause or has had a number of children.

If lutein cystomata are found, they should not be removed, for we know that they undergo spontaneous involution shortly after removal or expulsion of the mole.

After discharge from the hospital, these patients should be carefully observed because of the possible occurrence of chorionepithelioma. For many years it was taught that any woman who expelled a mole should be watched carefully for some time, and if hemorrhage or any anomalous bleeding made its appearance, the uterus was curetted and the scrapings subjected to miscroscopic examination by a competent pathologist. If chorionepithelioma was found, immediate hysterectomy with large doses of radium or *x*-ray was recommended. Cullen[11] and many others believe that a diagnosis of chorionepithelioma can not be made on curettings alone, and feel that a section of the uterine musculature must also be examined.

The Aschheim-Zondek (Friedman) test is of great value in the diagnosis of both hydatidiform mole and chorionepithelioma. While 1 c.c. of urine causes a positive reaction in normal pregnancy, the same may be obtained with $\frac{1}{50}$ c.c. in hydatidiform mole and with $\frac{1}{100}$ c.c. in chorionepithelioma, as has been reported by Castello.[12] Mazer,[13] Kimbrough, and others believe that the Aschheim-Zondek (Friedman) test should not be positive for longer than two weeks following a normal gestation, or eight weeks after the expulsion or

thorough operative removal of a mole. They feel that the presence of prolan in increasing concentration, longer than two weeks after a premature or full term pregnancy, is indicative of chorionepithelioma, and that the same is true when prolan persists for two months after a mole. Likewise, the reappearance of a positive test following one or more negative ones is pathognomonic of malignant degeneration.[22]

However, a case is reported by Falbusch,[20] in which the curettings led to a suspicion of malignancy. The Aschheim-Zondek test was negative; no operation was done, and the patient made a spontaneous recovery.

Balkow[14] states that the Aschheim-Zondek reaction is always positive in the presence of chorionepithelioma and considers it much more reliable than microscopic examination of the curettings. He feels that early diagnosis of chorionepithelioma by curettage is impossible. Leventhal and Sapir[15] observed that the Aschheim-Zondek test is a prognostic index in cases that have had a mole and, in the absence of clinical manifestations, the amount of gonadatropic substance in the urine in excess of 20,000 mouse units per liter indicates an early chorionepithelioma. Vozza[16] believes that the Aschheim-Zondek test should be done in all cases where chorionepithelioma is suspected. He feels that it is easier to establish a diagnosis by this means than by curettage.

Rosenstein[17] also shares this belief and suggests a course of procedure to be used in following cases that have expelled moles. He recommends that the test be done two weeks after the expulsion of a mole, and every two weeks thereafter until negative. It then should be repeated every four weeks for at least three months. He considers that a continued negative test during this time definitely rules out the presence of chorionepithelioma and notes that no exception to this statement is recorded. On the other hand, Schumann[24] recently reported a case in which curettings considered to be deciduitis were in reality chorionepithelioma. Two Aschheim-Zondek tests were negative. He feels that the biologic tests, especially with high dilutions of urine, in the presence of persistent fetal elements, makes the diagnosis of chorionepithelioma practically certain. He also believes that when, under the same conditions, these tests remain negative, one cannot be sure that chorionepithelioma of lower proliferation and malignant quality is not present.

Mathieu[23] warns that there is a 2 per cent error in the Aschheim-Zondek (Friedman) test. With the use of immature rabbits, a false negative is more probable than a false positive test. Therefore, a negative test which does not agree with the clinical picture might be false, and the test should be repeated. He does not believe a true negative Aschheim-Zondek test can be obtained in the presence of living chorionic tissue. He feels that if, in following cases of hydatidiform mole, one obtains two or three negative tests (enough to avoid the possibility of a false negative), he may be sure that chorionepithelioma will not develop from that mole or pregnancy.

Cases have been reported by Outerbridge,[18] Reis,[19] and others[22] in which long periods of time elapsed between the occurrence of pregnancy or hydatidiform mole and chorionepithelioma. These latent periods have varied from one and one-half to twenty years. One case is reported in a woman 75 years of age, twenty years past the menopause. In none of these cases was the Aschheim-Zondek test done. The question arises: Do these cases result from surviving chorionic elements in the uterus, or are they metastases from some other structure such as a teratoma of the ovary?

Mathieu[23] noted that 80 per cent of chorionepitheliomata were found within twenty days after the passage of the mole, in those cases in which the patient was properly examined, in which sufficient Aschheim-Zondek tests had been made, and in which pathologic specimens were examined. This fact, plus his clinical experience, leads him to the hypothesis that certain cases of mole have the potentiality of malignancy. In these patients the malignancy is present with the mole—

TABLE I

ANALYSIS OF CASES

LONG ISLAND COLLEGE HOSPITAL

HOSP. NO.	DATE	AGE	YRS. MARRIED	PREGNANCIES NO.	F. T.	ABOR.	L. M. P.	DURATION OF BLEEDING	MOS. SINCE LAST REG. PERIOD	SIZE OF UTERUS	HEMATOLOGY	ALBUMEN	BLOOD PRESSURE	OTHER SYMPTOMS	OPERATION: DIGITAL EVACUATION	VAG. HYSTEROTOMY	ABD. HYSTEROTOMY	HYSTERECTOMY	HOSPITAL DAYS	OVARIES CYSTIC	SUBSEQUENT HISTORY: PREGNANCIES NO.	F. T.	ABOR.	RECUR.	MENSES	LAST SEEN: YRS. AFTER OPERATION	CONDITION	A. Z.
4848 1 R.F.	1917	21	?		?		?	?	?	?	?	?	?	?	SPONT. EVAC.				18	-	3	3	0	0	NORMAL	17	GOOD	NEG.
2548 2 M.D.	1919	47	28	11	10	1	MAR. 1/19	8 WKS.	2½	4 MOS.	NOT DONE	TRACE	185/105	NONE				1	15	-	0	0	0	0	NONE	15	GOOD	NEG.
3198 3 R.L.	1919	40	24	5	5	0	JAN. 3/19	3 MOS.	6	3½MOS.	12200 W.B.C. Hb 75%	NEGATIVE	135/90	NONE	1				10	-	0	0	0	0	NORMAL	13	GOOD	NEG.
2582 4 P.S.	1919	23	5	0	0	0	OCT. 8/19	NONE	7	2½MOS.	10500 W.B.C. Hb 80%	NEGATIVE	104/60	NONE		1			11	-	4	4	0	0	NORMAL	13	GOOD	NEG.
2945 5 D.L.	1920	32	33	12	9	3	FEB. 1/20	4 MO.	5	5 MOS.	11500 W.B.C. Hb 80%	TRACE	200/100	NONE		1			17	-	0	0	0	0	NONE	10	KILLED IN AUTO ACCIDENT	
1220 6 R.C.	1921	25	5	2	2	0	DEC. 19/20	7 WKS.	2½	6½MOS.	9200 W.B.C. Hb 88%	TRACE	110/70	ABDOMINAL ENLARGEMENT		1			19	-	2	2	0	0	NORMAL	14	GOOD	NEG.
4177 7 H.S.	1921	23	4	1	1	0	JULY 28/21	4 WKS.	2	4 MOS.	7800 W.B.C. Hb 78%	4+ 5 GRAMS TO LITER	146/98	HYPEREMESIS TOXEMIA	1				12	-	2	2	0	0	NORMAL	14	GOOD	NEG.
4706 8 M.M.	1921	36	8	2	2	0	JULY 1/21	3½MOS.	3½	3½MOS.	12200 W.B.C. Hb 88%	4+ 8 GRAMS TO LITER	145/85	TOXEMIA			1		25	-	0	0	0	0	NORMAL	14	GOOD	NEG.
498 9 F.E.	1925	24	1	0	0	0	NOV. 14/24	2 MOS.	2½	4 MOS.	6800 W.B.C. Hb 84%	TRACE	100/60	NONE		1			20	-	0	0	0	0	NORMAL	9	GOOD	NEG.
1479 10 M.R.	1925	32	7	3	3	0	JULY 29/24	2 MOS.	7	4 MOS.	7200 W.B.C. Hb 94%	NEGATIVE	120/70	NONE		4			47	-	2	2	0	0	NORMAL	9	GOOD	NEG.
8796 11 A.S.	1925	32	6	4	4	0	OCT. 11/25	2 MOS.	2½	4 MOS.	12000 W.B.C. Hb 52%	TRACE	130/80	LOWER ABD. PAIN	1				28	+	3	3	0	0	NORMAL	9	GOOD	NEG.
1938 12 R.W.	1926	44	22	3	2	1	OCT. 21/25	1 MO.	4	5 MOS.	12400 W.B.C. Hb 68%	TRACE	144/84	NONE	1 WITH RADIUM				25	-	0	0	0	0	NONE	8	GOOD	NEG.

TABLE II

LONG ISLAND COLLEGE HOSPITAL

HOSP. NO.	DATE	AGE	YRS. MARRIED	PREGNANCIES NO.	F. T.	ABOR.	L. M. P.	DURATION OF BLEEDING	MOS. SINCE LAST REG. PERIOD	SIZE OF UTERUS	HEMATOLOGY	ALBUMIN	BLOOD PRESSURE	OTHER SYMPTOMS	OPERATION: DIGITAL EVACUATION	VAG. HYSTEROTOMY	ABD. HYSTEROTOMY	HYSTERECTOMY	HOSPITAL DAYS	OVARIES CYSTIC	SUBSEQUENT HISTORY: PREGNANCIES NO.	F. T.	ABOR.	RECUR.	MENSES	LAST SEEN: YRS. AFTER OPERATION	CONDITION	A. Z.
5572 13 C.V.	1927	34	6	3	3	0	MAR. 11/27	1 MO.	4	5 MOS.	11800 W.B.C. Hb 60%	TRACE	112/80	LOWER ABD. PAIN	1				9	-	0	0	0	0	NORMAL	7½	GOOD	NEG.
1418 14 A.R.	1928	30	13	2	0	2	OCT. 20/27	1 MO.	4	3½ MOS.	13900 W.B.C. Hb 70%	NEGATIVE	110/70	LOWER ABD. PAIN		1			21	+	0	0	0	0	NORMAL	8½	GOOD	NEG.
4763 15 C.L.	1928	23	1	0	0	0	JULY 20/27	4 MOS.	9	3 MOS.	9200 W.B.C. Hb 80%	NEGATIVE	113/85	NONE		1			18	-	2	2	0	0	NORMAL	8½	GOOD	NEG.
7255 16 M.C.	1931	30	11	3	3	0	JUNE 15/31	3 MOS.	3½	4 MOS.	6230 W.B.C. Hb 82%	NEGATIVE	100/80	NONE	SPONT. EVAC.				10	-	0	0	0	0	NORMAL	3	GOOD	NEG.
6484 17 E.G.	1932	24	4	1	1	0	MAR. 1/32	3 MOS.	6	3 MOS.	11850 W.B.C. Hb 79%	NEGATIVE	110/72	NONE		1			12	-	0	0	0	0	NORMAL	3½	GOOD	NEG.
582 18 A.C.	1932	25	7	4	4	0	SEPT. 24/31	4 MOS.	4	4 MOS.	9400 W.B.C. Hb 65%	NEGATIVE	110/85	LOWER ABD. PAIN	SPONT. EVAC.				17	-	1	1	0	0	NORMAL	2	GOOD	NEG.
2011 19 J.S.	1933	31	3	0	0	0	JAN. 14/33	NONE	3½	5 MOS.	Hb 90%	4+ 10 GRAMS	190/100	TOXEMIA	1				7	-	2	2	0	0	NORMAL	2	GOOD	NEG.
4422 20 S.D.	1933	35	12	4	2	2	APRIL 25/33	2-MOS.	3	4 MOS.	12600 W.B.C. Hb 68%	TRACE	120/78	NONE	1				13	-	1	1	0	0	NORMAL	1½	GOOD	NEG.
4520 21 C.L.	1934	43	16	15	15	1	OCT. 28/33	2 MOS.	2	2 MOS.	15000 W.B.C. Hb 78%	NEGATIVE	130/90	NONE	1	8	1	1	11	-	0	0	0	0	NORMAL	3½	GOOD	NEG.
3803 22 A.M.	1935	27	1	0	0	0	FEB. 19/35	3 MOS.	3	8 WKS.	5050 W.B.C. Hb 84%	NEGATIVE	108/88	HYPEREMESIS	1				14	-	0	0	0	0	NORMAL	2	GOOD	NEG.
5942 23 A.K.	1936	35	8	3	0	3	MAR. 28/36	1 MO.	8	5 MOS.	10000 W.B.C. Hb 70%	NEGATIVE	120/70	HYPEREMESIS	1				5-5-6	-	0	0	0		NORMAL	6 MOS.	GOOD	NEG.
3755 24 E.R.	1937	26	2	1	0	0	DEC. 5/36	2 MO.	4	5 MOS.	7400 W.B.C. Hb 77%	NEGATIVE	110/80	NONE	1				10	—	0	0	0	0	NORMAL	4 MOS.	GOOD	NEG.

actual malignant changes have already taken place and even though the mole is passed or removed, the malignant cells remain.

During the twenty years from January 1, 1917, to January 1, 1937, twenty-four cases of hydatidiform mole were admitted to the gynecologic service of the Long Island College Hospital.

TABLE III
AGE INCIDENCE 21 TO 52

Years	Cases	Per Cent
20–30	10	41½
30–40	10	41½
Over 40	4	16½

PARITY

Parity	Cases	Per Cent
Multipara	17	71
Nullipara	6	25
Not noted	1	4

TABLE IV
ONSET OF FIRST SYMPTOMS

Months	Cases	Per Cent
2	3	75
2½	5	
3	2	
3½	3	
4	4	
5	1	25
6	2	
7	2	
9	1	
Not noted	1	

We have been unusually fortunate in obtaining follow-up records on all of these patients, either by personal contact, or through other members of the hospital staff, who, as private physicians, were responsible for their care.

The follow-up data included the history as to general health, menses, and subsequent pregnancies. In addition, the Aschheim-Zondek (Friedman) test was done on the urine of every patient but one (this patient was killed in an automobile accident ten years after expelling a mole).

TABLE V

URINALYSIS	
Albumin marked	3
Albumin trace	8
Albumin negative	11
Albumin not noted	2
HYPERTENSION	
Present	9
Not present	14
B/P not noted	1

TABLE VI
SIZE OF UTERUS IN RELATION TO PERIOD OF PREGNANCY

Size	Cases	Per Cent
Larger	11	45
Smaller	8	33⅓
Normal	4	16⅔
Not stated	1	5

TABLE VII
SYMPTOMS OTHER THAN BLEEDING

Marked abdominal enlargement	1
Hyperemesis	3
Toxemia	3
Lower abdominal pain	4

TABLE VIII
TREATMENT

Spontaneous evacuation	3
Digital removal	10
Digital removal with radium	1
Anterior vaginal hysterotomy	8
Abdominal hysterotomy	1
Abdominal hysterectomy	1

TABLE IX
SUBSEQUENT PREGNANCIES

Cases sterilized or near menopause	7
Cases in whom pregnancy was possible	17

Of these 17 cases 9, or 53 per cent, became pregnant and 8, or 47 per cent, did not conceive.

TABLE X
MORBIDITY AND MORTALITY

Morbidity (using as a standard 100.4°F. on two consecutive days excluding the first 24 hours)	0
Maternal mortality—primary	0
Maternal mortality—remote	0

SUMMARY OF FINDINGS

1. An analysis of twenty-four cases of hydatidiform mole is presented.

2. Twenty patients (83 per cent) were under forty years of age.

3. Seventeen patients (71 per cent) were multiparae.

4. Three patients (12½ per cent) had as initial symptoms, evidence of fulminating pre-eclamptic type toxemia, with marked edema, hypertension and albuminuria. It is noteworthy that the signs of toxemia were established before the end of the first trimester of pregnancy.

5. The uterus was enlarged out of proportion to the period of gestation in only eleven cases (45 per cent). However, all eleven cases were noted in women who were examined before the end of the fourth month of pregnancy. Patients examined after the fourth month showed the uterus to be about normal size or smaller than normal for that period of gestation.

6. Only two cases (8 per cent) showed cystic enlargement of the ovaries.

7. Twenty-two cases (over 91 per cent) were treated from below, while only two cases (8 per cent) were treated from above, both as a result of error in diagnosis. In one instance an abdominal hysterectomy was performed on a patient 47 years of age with a diagnosis of fibroid uterus. In the other case abdominal hysterotomy was performed for fulminating toxemia that did not respond to treatment, and in which hydatidiform mole was not suspected.

8. The good results following treatment from below seem to indicate that this method of approach is preferable to laparotomy, as suggested by Schumann and others.

9. One patient received 3,000 mg. hours of radium after digital removal of a partially retained mole. She was 44 years of age and had been treated in another institution six weeks prior to admission to the Long Island College Hospital.

10. Seventeen patients (53 per cent) subsequently became pregnant and were delivered of full-term normal infants. There was no recurrence of mole in any of them.

11. There was no morbidity in this series.

12. There was no mortality, either primary or remote.

13. Chorionepithelioma did not develop in any of these cases and Aschheim-Zondek (Friedman) tests done on the urine long after the expulsion or removal of the mole, failed to show evidence of surviving chorionic elements.

14. While this small series of cases undoubtedly should not be interpreted as a criterion of actual incidence, nevertheless, it seems to bear out the contention of Novak, Black, and others that hydatidiform mole is not followed by chorionepithelioma as frequently as is commonly thought.

15. It is understood that in advocating the management of these cases per vaginam, we still consider that the patient is to be observed for the occurrence of irregular bleeding and that the Aschheim-Zondek (Friedman) tests are to be repeated frequently during the first year following the expulsion or removal of a mole.

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GRANULOMA INGUINALE (VENEREUM) IN THE FEMALE*

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Definition. Granuloma inguinale (venereum) is a chronic specific infection chiefly of the skin and subcutaneous tissues, and in the female primarily involves the external genitalia or the inguinal region. Less frequently the vagina or the cervix is the primary site of the disease. The causal organism is believed to be the Donovan body. The disease is probably venereal in origin and occurs most commonly in the colored race.

Etiology. It is generally accepted that the Donovan body is the causative agent. Several investigators, however, believe it is due to a bacillus belonging to the mucosus capsulatus group of Friedlander. McIntosh[1] claims to have grown this organism on various ordinary culture media, but this could not be substantiated by Dienst, Greenblatt and Sanderson[2] who have been unable to cultivate the Donovan bodies in spite of the fact that on several occasions they fortunately obtained the organism free from any type of bacteria from unruptured pseudo-buboes. They are convinced that the organism is not a bacterium. De Monbreun and Goodpasture[3] recovered the Donovan bodies from artificially infected monkeys. One of us (R. B. G.) has succeeded in transmitting the disease from one patient to another with recovery of the organism in a pure state from the inoculated person one month and two months afterwards.

The disease is thought to be venereal because the primary lesion most often appears on the genitalia, and only during the period of active sex life of its victims. Individual susceptibility, uncleanliness and race are important etiologic factors. It is possible that the disease may be transmitted by some insect vector, but this has never been proved.

Pathology. Pund and Greenblatt[4] summarize the pathology thus: "Granuloma venereum reveals in the pure or unmixed cases a uniform histologic picture. The essential features are (*a*) the massiveness of the cellular reaction in which the luxuriant granulation tissue is surcharged with plasma cells; (*b*) the relative and conspicuous paucity of lymphocytes; (*c*) the diffuse sprinkling of polymorphonuclear leucocytes, with focal collections in the superficies and papillae; (*d*) the pronounced marginal epithelial proliferation simulating early epitheliomatous changes; and (*e*) the pathognomonic large mononuclear cells scattered in various numbers throughout the granulation tissue. (Fig. 1.)

"The pathognomonic cell is specific for granuloma venereum. The relatively large size of the cell, the diameter of which varies from 25 to 90 microns, and the many intracytoplasmic cysts filled with deeply stained bodies are its cardinal features. Donovan bodies are round or rod-like and are grouped peripherally within the cysts

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and have an affinity for hematoxylin. (Fig. 1.) Their recognition is of paramount importance, for it permits the diagnosis of

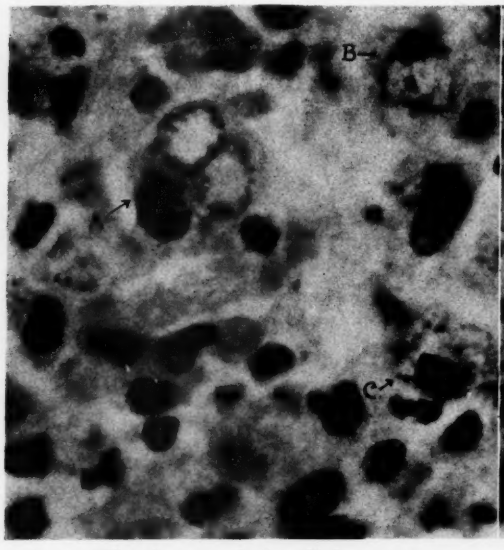


FIG. 1. The characteristic cellular response as observed in tissue section stained with hematoxylin and eosin. A, the pathognomonic cell is diagnostic of granuloma inguinale. Note the small rounded bodies lining the periphery of the intracytoplasmic cysts. B and C, pathognomonic cells slightly out of focus. × 2250.

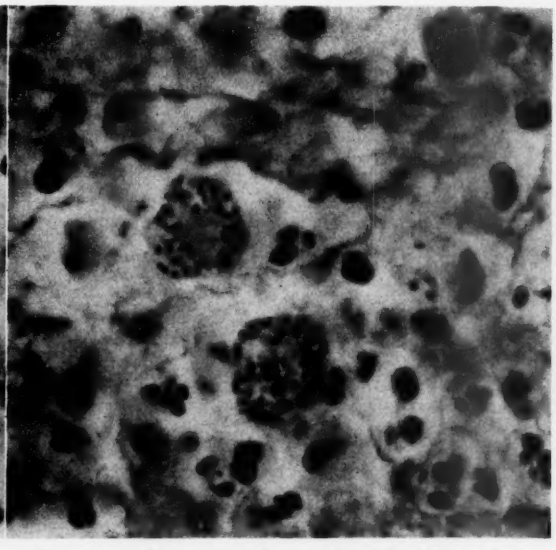


FIG. 2. A biopsy section stained by Dieterle's silver technique. Note the characteristic (closed) safety pin appearance of some of the so-called Donovan bodies filling the intracystic spaces of the pathognomonic cells. × 2250.

granuloma venereum to be made by histologic study of the tissue."

To this description the authors[5] have further added that: "The affinity of the intracystic bodies for silver salts facilitates the recognition of the characteristic cell. With silver these bodies are stained black to brown and have a (closed) safety pin appearance because of their elongated ovoid and intense bipolar staining reaction." (Fig. 2.)

The rôle played by the lymphatics in this disease is at present under study. Histologic study of tissue removed from the inguinal pseudo-bubo reveals not a lymphadenitis, but a subcutaneous granulomatous reaction.

Clinical Manifestations. In the past the literature and textbooks have pictured this disease as an extensive, destructive, incapacitating ulceration, foul to one's nostrils and loathsome of one's sight. An understanding of the early lesions will dispel such a notion. The recognition of its various manifestations in this phase is essential, for it is quite amenable to treatment and cure. A knowledge of the early vulvar, perineal, vaginal, cervical and inguinal types of lesions is therefore of primary importance. A typical early vulvar lesion is illustrated in Figure 3. The clean raised tuft of velvety red granulation tissue with well defined borders is characteristic. A small perineal lesion, slightly ulcerated in its central portion but with definite rolled edges, is demonstrated in Figure 4. The vaginal manifestation exhibiting the raised rolled margin is pictured in Figure 5. The purely cervical nature of this disease, which has been only recently established[5] as belonging to this group, is reproduced in Figure 6 from a drawing of a characteristic case. Figure 7 illustrates an inguinal subcutaneous granuloma (pseudo-bubo) shortly after ulceration. Before ulcerating it appeared as an indurated mass which clinically did not differ greatly from the bubo of lymphogranuloma venereum or that of chancroid.

The early lesion, if it remains undiagnosed or if treatment is delayed, may spread to involve the whole pudendal re-

gion. The lesions soon become secondarily infected with spirochetes, fusospirochetes and/or other organisms. The ulcerations

Fig. 3. Early vulvar granuloma inguinale (venereum).

Fig. 4. Early granuloma inguinale (venereum) of perineum.

then become extensive, deep, destructive and foul. Figures 8 and 9 illustrate such a case.

In addition to the above manifestations, granuloma inguinale (venereum) has been found extragenitally (upper lip in one case[6] and neck in another[11]). In one case reported by Pund and Gotcher[7] the body of the uterus, the tubes and ovaries were extensively involved.

Complications. Syphilis, or evidence suggestive of syphilis to the extent of a positive serologic test, is the most common complication. Important as it is to treat the syphilis, it has proved a bugbear in masking the true diagnosis. Another common complication altering the nature of the disease and adding to its virulence and chronicity is secondary infection with fusospirochetes. (Fig. 10.) Coincident lymphogranuloma venereum in its various forms, such as elephantiasis vulvae or rectal stricture, as well as chancroid ulceration or

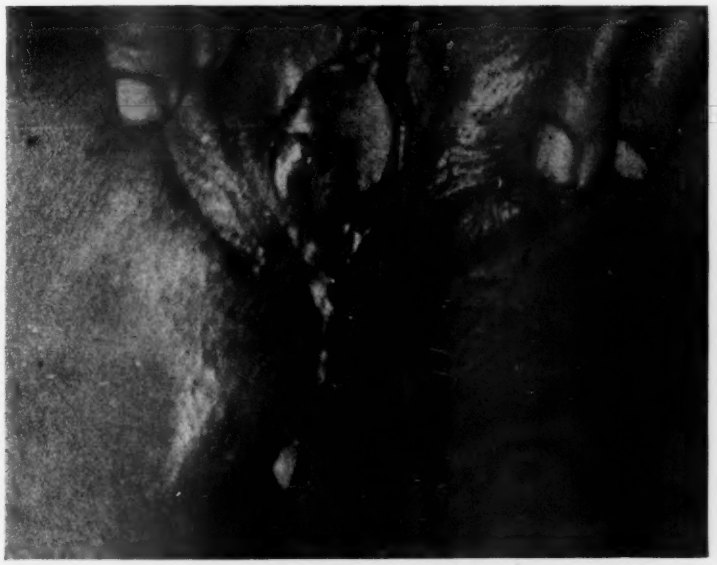


Fig. 5.—Early vaginal granuloma inguinale (venereum).

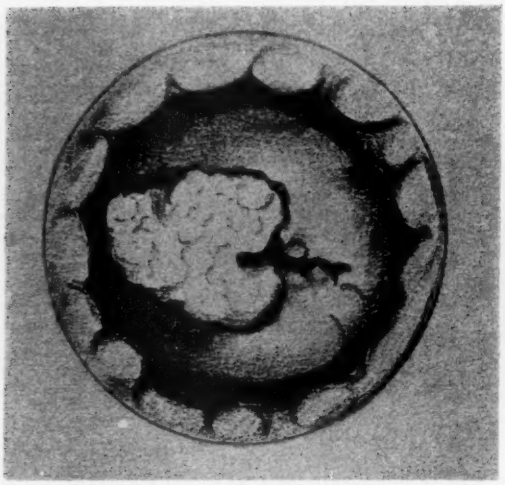


Fig. 6. Cervical granuloma inguinale (venereum). (Photograph from a drawing by Dr. E. A. Wilcox, University of Georgia School of Medicine.)

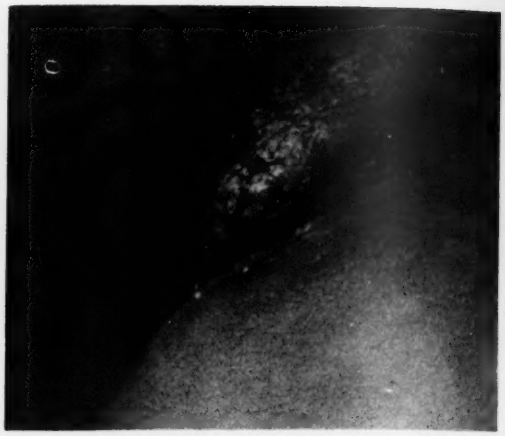
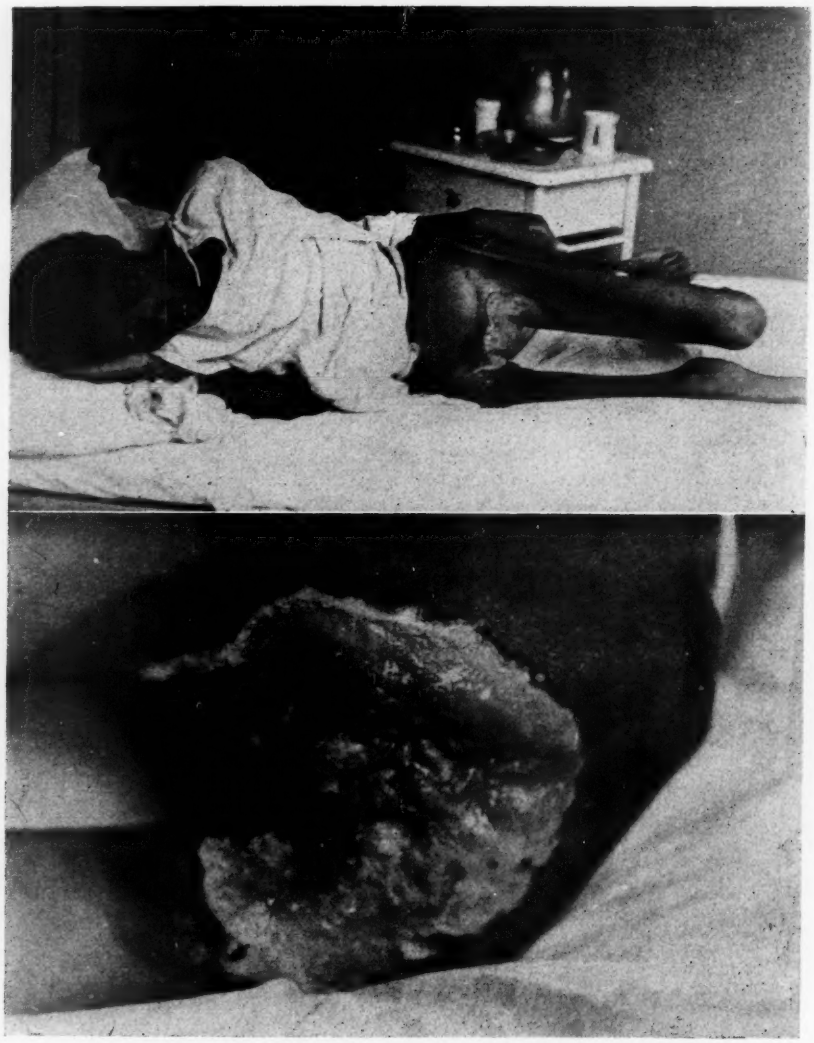


Fig. 7. Pseudo-bubo of granuloma inguinale (venereum).



Figs. 8 and 9. Advanced granuloma inguinale (venereum).

gonorrhea, may be present. In fact all five of the venereal diseases have been observed in one and the same patient.

unhealthy appearance with ropy puriform discharge. It must be differentiated from all other causes of chronic granulomatous

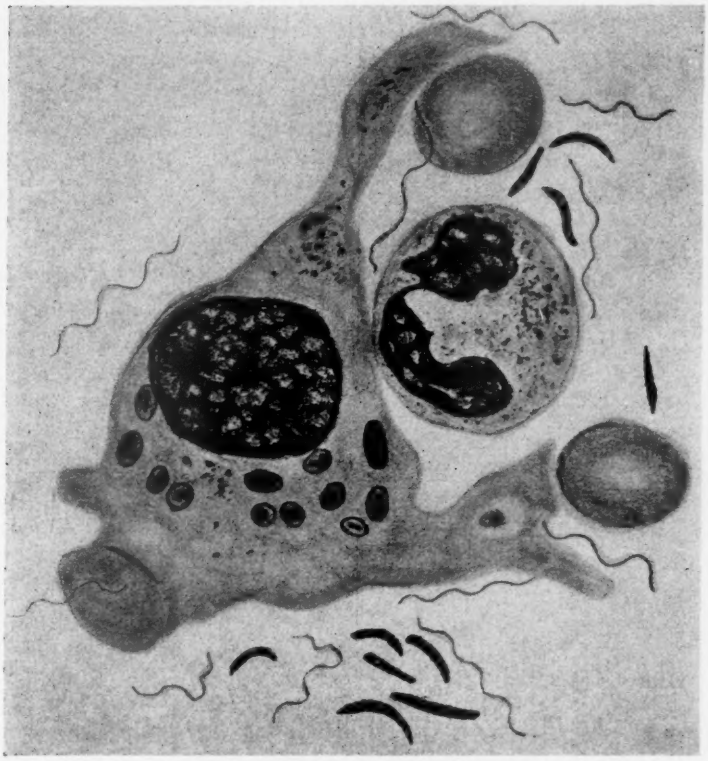


Fig. 10. Photograph of a camera lucida drawing × 2000 of Donovan bodies within histiocyte. With Wright-Giemsa stain, capsules of the bodies are pink, metachromatic bars or nuclei are blue-black. Note superimposed fusospirochetal infection to which usually insufficient attention is given. (From Greenblatt et al., in *J. M. A. Georgia, 26:* 16, 1937.)

Diagnosis. It is to be borne in mind that granuloma inguinale (venereum) is a chronic infection usually limited to the skin and subcutaneous tissues and not primarily of the lymphatics. However, it has been found in the vagina and on the cervix. The affection is characterized by a chronic ulcerative, destructive granulation tissue that commences from a small macule somewhere in the pudendal region. It grows by continuity and may spread from the groin or perineum to the abdomen and thighs. Early the granulations are pink to red in color and are raised and clean in appearance. At this stage the granulations bleed readily on mechanical irritation. After secondary infection sets in, it may be covered by yellowish crusts and takes on an ulceration, especially lymphogranuloma venereum, chronic chancroid and fusospirochetosis, as well as secondary forms of syphilis, carcinoma and tuberculosis.

The clinical diagnosis of granuloma inguinale (venereum) must be corroborated by the demonstration of Donovan bodies in smear or in tissue section. A positive diagnosis should be made only when these bodies are found.

While the uncomplicated disease is fairly uniform in its manifestations, the incidence of associated secondary contaminants is so high that the clinical picture is frequently altered. A successful way fully to diagnose each case is to carry out the following routine, much of which is laboratory in nature: (1) repeated Wassermann or Kahn

tests to rule out syphilis; nigrosine stain[8] or dark field study for treponema pallidum and other spirochetes; (2) smears stained for gonococci, Donovan bodies, fusospirochetes, Ducrey bacilli, yeasts, etc.; (3) chancroid intradermal tests;[9] (4) Frei test; (5) biopsy for Donovan bodies in tissue section, since not infrequently it is difficult to demonstrate these bodies in smears because of marked contamination by other organisms. The biopsy will establish the diagnosis in such cases and at the same time rule out tuberculosis, carcinoma, etc. To facilitate the diagnosis, all buboes, including the pseudo-bubo of this condition, should be aspirated under sterile conditions and routine smears and cultures made.

Therapy. I. The complete diagnosis with the complications is the first requisite.

II. Treat syphilis, gonorrhea or chancroid, if present, coördinately.

III. Treat fusospirochetosis or other invaders when present. These are often the cause of much discomfort and great damage to tissues. Specific therapy of granuloma inguinale (venereum) has little effect unless these secondary invaders are checked. We have found 4.5 per cent neoarsphenamine in equal parts of glycerine and cod liver oil excellent as a local application to check the phagedenic effect of the saprophitic fusospirochetes, to serve as a prophylactic against such contaminants and to promote healthy granulations and healing.[10] This mixture will prove more tolerable if some local analgesic such as nupercaine is added. Perborate, peroxide or potassium permanganate washes are valuable.

IV. Specific treatment with fuadin intramuscularly or 1 per cent tartar emetic intravenously is of definite value, notably in the early cases. This therapy must be continued over a long period of time even after apparent cure. If this is not done recurrences are inevitable. The value of this medication as a therapeutic agent decreases in direct ratio with the chronicity and duration of the disease. *Therefore, early diagnosis of the lesion is of the greatest importance.*

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TENDON INJURIES

THEIR CLASSIFICATION AND EARLY TREATMENT

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Introduction. Of all the injuries to the hand—that incomparable tool of the human machine—injuries to the tendons and nerves, and infections of the tendon sheaths, are often the most difficult to manage, the most protracted in their convalescence, and the most likely to leave a permanent disability. A derivative of connective tissue, tendon has inherited in a measure its ancestral capacity for regeneration, but has lost its hardihood towards infection to such an extent that it is safe to assume that any tendon that lies completely bathed in pus for more than forty-eight hours may lose its viability and slough out. Exposed to vulnerability in the fingers and hand, and susceptible of infection with possible permanent damage and deformity, tendons, when injured, become a major surgical problem economically, psychologically, and socialogically.

Classification. In considering tendon injuries, one is impressed by the close similarity in the mechanism operating in producing these traumapathies to that of bone injuries, and their treatment may well be approached along the same lines as in the case of fractures. Thus considered, tendon injuries fall into two types: (1) simple, without any break in the skin; and (2) compound, in which the laceration in the tendon follows a break in the skin and underlying tissues, exposing it to contamination. Either may be complete or incomplete; retracted (displaced) or not retracted; single or multiple; comminuted (frayed) or non-comminuted.

Type 1 is often the result of an injury from a blunt instrument, as at the knuckles where the taut tendon is caught between the traumatizing agent and the bone, and breaks or tears. It may also follow muscular violence, as in a rupture of the quadriceps extensor tendon, plantaris tendon, or the extensor tendons of the fingers from their insertion, etc.

Type 2 follows an injury from a sharp penetrating instrument, such as a knife, glass, scissors, etc.

Viewed in this light, we approach the treatment of lacerated tendons, which are compounded, in the same manner as we do compound fractures. Appreciating that the time interval for the application of treatment is an important element in the ultimate successful outcome, we treat these cases as an emergency, knowing that the earlier the reduction, the easier and better it will be. The retracted ends of the tendon are more readily coapted without any difficulty. Experience teaches us that here, as in all wounds of compounding, there is a golden period which lies within the first six hours of the injury, during which time the wound is contaminated but not infected, and any operative procedure accomplished within this time is more likely to result in primary union and obviate the possibility of secondary infection. Also, whenever infection has already set in, it is best to treat it first, resorting to secondary repair or a plastic reconstruction of the tendons six months to one year later when we are reasonably sure that a latent infection will not be stirred up by operative intervention.

We appreciate that lacerated tendons without a break in the continuity of the skin may be difficult to diagnose, attention more often being centered upon the effects of the contusion of the surrounding soft and hard tissues. The true lesion is not dis-

covered until later, when persisting impaired function indicates the possibility of a tendon injury. It is noteworthy that here again the analogy to a fracture can be further considered, in that sometimes we have an unrecognizable linear fracture in which the attention is drawn to the contusion of the soft parts, the fracture being revealed only by a routine *x*-ray check-up.

Healing in tendon injuries also exhibits a close similarity to that of bone, in that both show early callus formation, to be followed later by replacement of tendon and bone tissue, respectively. The paucity of contact tissue functioning in the repair of tendon—the cross section of which is but a small fraction of its entire length—and the great strain placed upon the tendon in the action of the muscle, bear another similarity to bone, where the cross section of the healing fracture site is but a small part of the entire length of the bone, yet the weight-bearing and supportive stress placed upon it may be very great.

Repair. The scanty vascular supply of tendon in general, and sheathed tendons in particular, and the necessity of a good vascular supply for repair, are two factors which appear to be constantly working against the surgeon. The clinical observation that unsheathed tendons which receive a liberal vascular supply from surrounding areolar tissues are more resistant to infection and heal better and more quickly than their sheathed kindred, is amply demonstrated in the rapidity and persistent regularity with which the severed tendo Achillis heals, even in the presence of a decided gaping.

The normal function of the tendon sheath is to act as a fluid buffer where tendon rubs against bone or ligament (Mayer[1]). This is particularly true whenever a tendon, at some phase of its motion, is forced to turn a corner. The rôle of the tendon sheath in repair of a severed tendon is to establish early continuity within a few days. Mason and Shearon[2] have shown that after the fourth or fifth day the tendon itself begins to proliferate and to send cells into the callus, and if the gap is not too big it may be bridged by tendon cells within about two weeks. After the sheath has served its purpose of splinting the early union, it begins to become more lax and areolar and, in a successful suture, to take on again its original function of serving as gliding tissue. This process of healing also takes place in autogeneous tendon grafts, as well as sutured tendons. The force of these facts is more strongly brought out when it is remembered that adequate vascularization of grafted tissues, e.g., skin or bone graft, require rest and immobilization necessary to obtain an efficient vascular bed to secure union, while rest and immobilization of injured tendons or in tendon grafts favor the formation of adhesions—the most important single factor making for the functional failure of operation on tendons. Experimental and clinical observations have shown that function is of major importance in determining the histologic structure of tendon tissues and emphasizes the necessity for purposeful movement of tendons during the course of healing.

The tension at which tendons should be sutured is a very difficult and perplexing problem. While there is no doubt that freshly severed tendons should be sutured end-to-end, after relaxation has been obtained by bringing the origin and insertion of the muscle as close together as possible, the problem is not so simple when one considers suture and repair of old injuries. Here the retraction is due not only to muscular tonus, but to intramuscular fibrosis as well, and while the former may be overcome, the latter is, to a certain extent, permanent, and end-to-end suture leads to permanent shortening of the tendon. This necessitates some form of plastic tendon lengthening to overcome the post-fibrotic shortening.

Pathology. The fundamental pathology occurring in tendon injuries follows a single local damage to the skin. But there is a wide diversity of involvement of the underlying structure so that often a

seemingly harmless laceration of the skin may reveal, upon exploration, a multiplicity of severed tendons, nerves, and

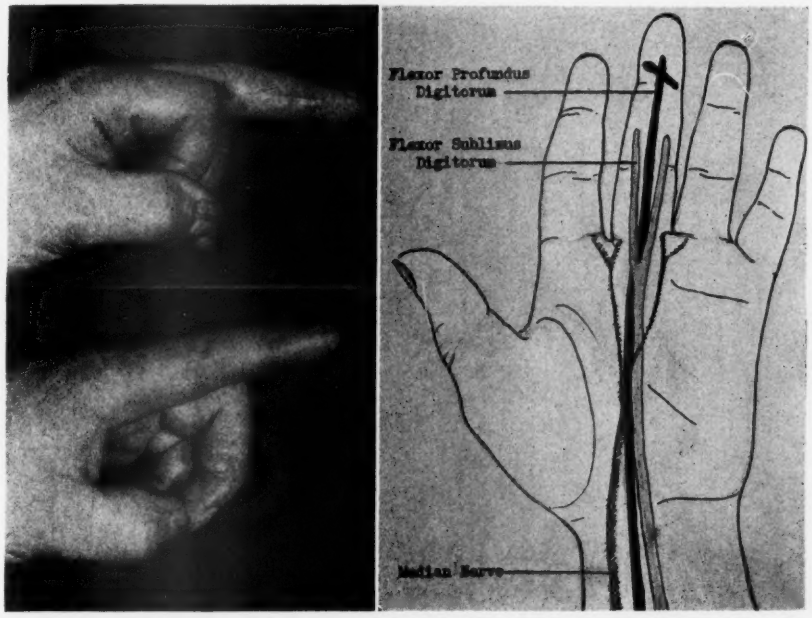

Fig. 1. Laceration across flexion crease of distal interphalangeal joint of middle finger, left hand. Operative repair. Result ten weeks after operation showing lateral prolongation of operative incision in search for proximal end of tendon.

blood vessels. These injuries are not alone localized to the site of injury but extend above and below its level, depending upon the state of tonus or tension of the individual tendons and their corresponding muscles at the time of the injury, and the movements and position of the hand at the time of the impact. This becomes especially apparent in injuries on the flexor surface of the wrist. There the flexors of the fingers and the nerves to the hand and fingers form the greatest single convergence in the body of a number of important tendons and nerves, all of which are exposed to vulnerability by a single stab or cut. The crowding of all these important structures into a small space makes for a great tendency to agglutination by adhesions following injury.

The wrist region, both flexor and extensor, because it has traversing about it all the tendons (twenty-six) and nerves to the hand and fingers, becomes the most vulnerable site for tendon damage. Clinically, this level shows a high incidence of tendon injuries. Unfortunately, the surface of the tendon most exposed to friction and the one most prone to adhesions—the anterior surface—receives the greatest effect of the impact in complete severance and is the one injured when partial injury of the tendon occurs.

Similarly, a large tendon callus follows laceration of the extensor tendons over the knuckles, appearing on the superficial or exposed surfaces of these tendons. Retraction is greater in flexor tendons than in extensor tendons, with the exception of extensors to the thumb where the proximal ends are often difficult to find because of a rapid and extensive retraction, sometimes as much as two-thirds up the forearm. The extensor tendons of the fingers, because of the lateral expansions, remain more fixed when severed, and do not retract as much as the flexor tendons.

The writer has found that the nearer the injury of the tendon to its insertion, the greater the retraction, so that the greatest retraction is found in lacerations of the flexor tendons in the fingers (espe-

cially the little finger) and palm of the hand, and lacerations of extensor tendons to the thumb at its base. However, the greatest single factor responsible for retraction following severance of a tendon is the time interval elapsing from the injury to surgical intervention. Where retraction is already established, finding the tendon may be done by milking the proximal end of the tendon distalward, following relaxation of the involved muscles, or by catheterizing the sheath with a probe until the proximal end is felt, the tendon located, transfixed, and threaded through its sheath toward the operating field.

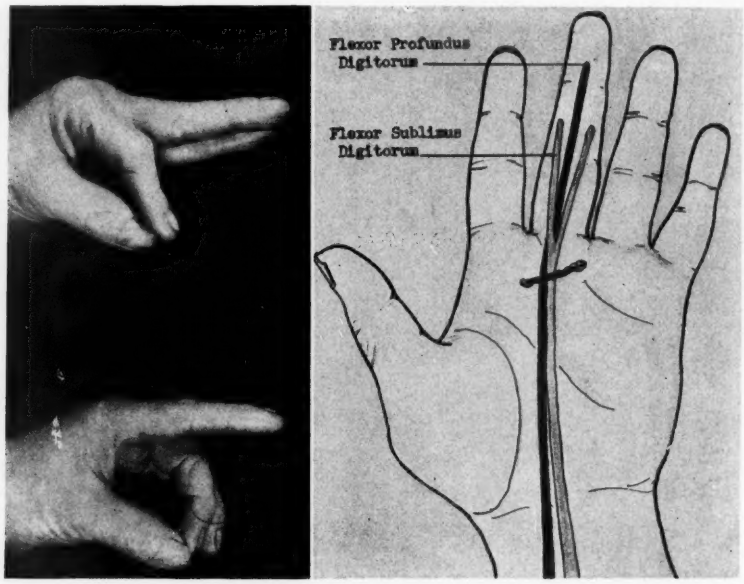

FIG. 2. Laceration of palm of hand at base of middle finger involving flexor profundus and flexor sublimis tendon before bifurcation. Suture of tendons. Result three months after operation.

Treatment. As stated, treatment in tendon lacerations is based on the type of injury present. In Type 1 injuries, where there is a laceration or rupture of tendons without break in the skin, we are dealing with a clean injury without contamination, and any exposure of the tendons is made with surgical intent under aseptic conditions predisposing to uniform primary healing. It is possible, in selected cases of this type such as avulsions of the extensor tendons of the fingers from their insertion or laceration of these tendons near their insertion, sometimes to effect repair without operative intervention by maintaining the finger in hyperextension by means of some adequate splintage.

We agree with Mason[3] that healing is more apt to take place in avulsed extensor tendons, when there is a flake cortical fracture of the phalanx associated with the avulsion by maintaining the finger in slight hyperextension for about six weeks. The regenerative power of the extensor tendons is such that if given a chance by good approximation, healing will take place. It must be borne in mind, however, that under these conditions healing may occur with the tendon in a lengthened condition with subsequent dropped finger tip. Also, in most tendon injuries occurring near the insertion of the extensor tendons, the joint space of the distal interphalangeal joint is opened, allowing loose tabs of tissue to lie within the joint, predisposing to ankylosis in partial flexion. Because of these possibilities, operative repair becomes the procedure of choice. If the joint space is cleaned out of loose or attached tabs of tissue, the capsule and tendon carefully repaired with fine silk, hyperextension maintained for three weeks, and no infection incurred, the results are

usually good. All other subcutaneous tears or lacerations of tendons automatically fall into the operative group.

dressing. The patient is requested systematically to flex each phalanx of the individual fingers, beginning with the

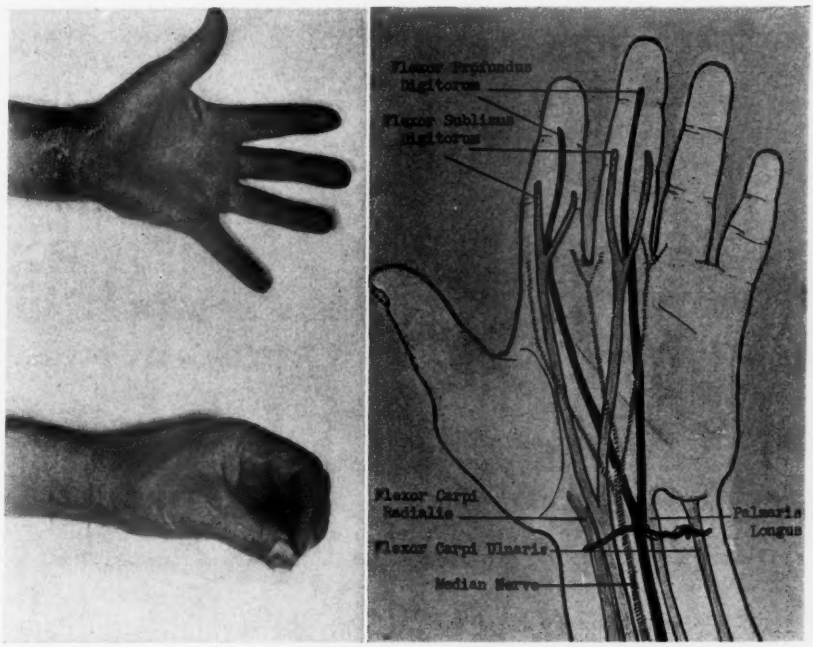

Fig. 3. Laceration across left wrist involving palmaris longus, flexor carpi radialis, flexor sublimis and profundus digitorum to index and middle fingers, flexor carpi ulnaris and median nerve. Immediate exploration and suture of tendons and nerve. Result five months after operation.

Type 2 injuries—or compound lacerations of tendons—are acute operative surgical emergencies, and are dealt with in the same manner as compound fractures. This implies an immediate sterilization of the wound, control of hemorrhage, and application of a sterile dressing. This is followed by temporary splintage of the part for complete relaxation of the origin and insertion of the tendons to minimize the amount of retraction that will take place until operative intervention. Hemorrhage, if present, has in our experience proved to be an excellent internal lavage of the wound, and it is the writer's observation that compound lacerations of the tendons in which a generous hemorrhage was present were more likely to heal without infection than lacerations of tendons in which the wound was bloodless, dry and anemic.

At this time the wound is looked at, but not into, and covered with a sterile

thumb, to determine the amount of tendon damage present, and whether tendons are partially or completely severed. Further, the integrity of the nerves to the parts is tested, by requesting the patient to use the intrinsic muscles of the hand, i.e., abduction and adduction of the fingers, and adduction of the thumb for the ulnar and apposition of the thumb for the median. The sensory involvement is also determined, giving a definite picture of the character and extent of the damage present and, to some degree, an index of the enormity of the operative problem.

As the operative procedure may be time consuming—especially where there is a multiplicity of tendons involved with crushing and contusion of lacerated tendon stumps—the type of anesthesia becomes important. General anesthesia is the one of choice. Local block anesthesia at the elbow is desirable wherever possible, as it allows the surgeon to gauge the tension

at which the tendons are to be sutured. This is particularly true in loss of tendon substance. Local infiltration at the site of injury is mentioned only to be condemned, as it further lowers the resistance of the already traumatized tissues and invites infection.

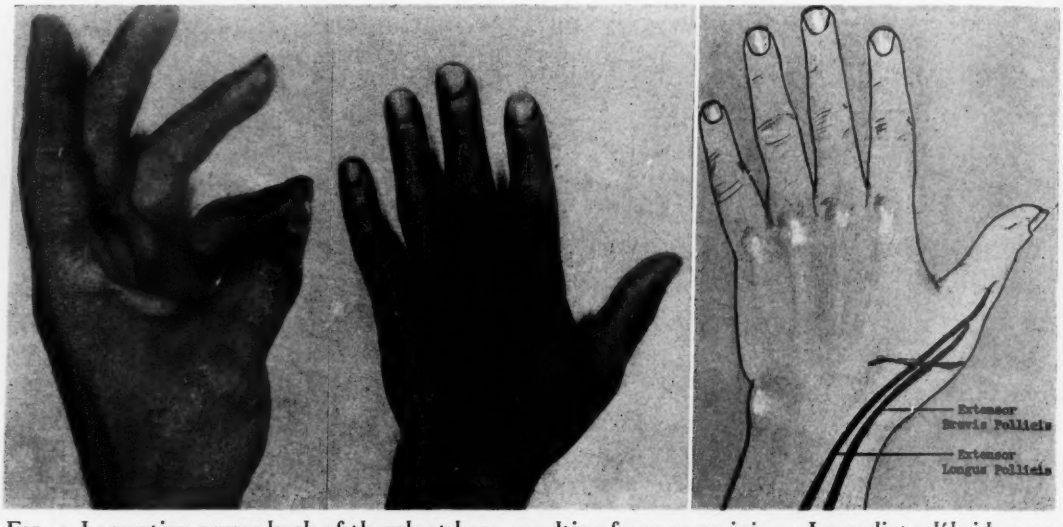

FIG. 4. Laceration across back of thumb at base, resulting from an ax injury. Immediate débridement and suture of the superficial and deep extensors of the thumb. Result fourteen weeks after injury.

Operative repair demands accurate end-to-end apposition of tendon stumps, using fine silk as the suture medium and the atraumatic lacing technique described by Bunnell.[4] This, however, should always follow a preliminary preparation of the skin and wound, by thorough scrubbing of the skin with soap and water. The wound is protected by a sterile sponge to prevent washing further contamination into it. The wound is then irrigated with copious amounts of sterile saline, thereby floating to the surface all débris, blood clots, or any foreign material that may have been introduced at the time of injury. The parts are dried, the skin painted with tincture of iodine, and the wound generously swabbed with the iodine. This is followed by instillation of half strength tincture of iodine into the wound itself. The iodine should reach every nook and cranny of the contaminated wound. This is essential to obviate the most important single complication that interferes with the success of the outcome in tendon repair, namely, *infection*.

Next, all dead and devitalized tissues from the surface to the depth of the wound are carefully excised, all structures being handled gently and carefully. One must avoid crushing tendons. Where necessary the frayed ends should be freshly cut to obtain good approximating surfaces. If it becomes necessary to extend the original wound for an adequate exposure in the operating field, it is important to remember to avoid cutting across the normal flexion creases in the digits, and, wherever possible, to refrain from cutting on the anterior surfaces of the fingers.

Searching for the retracted proximal ends of the flexor tendons in the fingers should be done by progressive incisions on the lateral surfaces, avoiding the flexion creases. When located, the end is transfixed with a silk suture, and with the finger and hand in flexion, the retracted end is gently threaded through the sheath and drawn into the operative field. As each proximal end of the tendon is located, transfixing it with a silk suture helps to identify it and minimizes the trauma produced by grasping and holding it with a clamp.

Where a loss of substance is present, the simplest and easiest type of tendon plastic

should be done for the restoration of length, avoiding the use of free tendon grafts in freshly divided tendons. Where a simple type of tendon plastic is not expedient, it is better to defer extensive tendon operation for a later reconstruction when the possibility of infection from a contaminating wound has passed. The deep flexor should always be saved when both superficial and deep flexors are involved, as it is the more powerful and more important of the two tendons. Repairing both at the same time tends to form adhesions between them, and between the tendons and the surrounding soft parts, unless provisions are made to prevent this. In loss of substance of both flexor tendons, the adjacent normal flexor sublimis may be transplanted and made to assume the role of main flexor for the involved finger.

Fine black suture is the one of choice in repairing freshly divided tendons because it can be easily handled without tearing through the tendon substance, produces the least absorptive reaction in the tissues, and allows early motion. It is important to use a straight non-cutting edge needle in order not to cut the tissues nor the suture when doubling up in the Bunnell[5] stitch. The knot is buried between the approximated surfaces.

Repair of the tendon sheath is an essential step because it protects the tendon suture line, prevents adhesions, and functions in the early stages of tendon repair. This is effected by using a continuous fine plain catgut suture. Where the tendon sheath is irreparably damaged, every effort should be made to reconstruct it, using a segment of vein (if necessary). The repaired tendon may be covered with fat, fascia, or areolar tissue before suturing the skin to prevent the formation of adhesions. The skin should be carefully approximated to insure primary union. It is important, when sewing the skin of the palm, to evert it so that it does not pucker in, causing adhesions between it and the palmar fascia. The use of a tourniquet is an advantage in operative tendon work, and the blood pressure machine cuff makes an excellent pneumatic tourniquet.

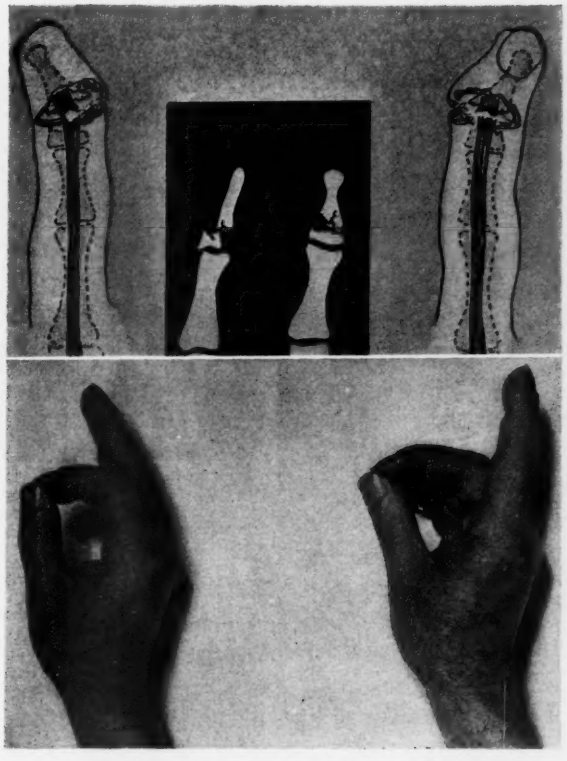

Fig. 5. Crushing injury of distal end of right index finger, with almost complete amputation of tip resulting in a complete laceration of the extensor tendon. Débridement, replacement of tip, and suture of skin only, and immobilization in neutral position without suture of tendons. Tendons were not sutured because at the time of operation it was felt that the small tab of skin bridging the tip to the rest of the finger might not be sufficient to maintain its circulation, but the tip remained viable and the tendons healed spontaneously. Functional result two months after operation.

When drainage is used, it should consist of a fine strand of twisted silkworm gut, with the drain placed to the tendon sheath and not within it, that is, drainage is for the skin and subcutaneous areas. Maintenance of relaxation at the operative site to relieve tension on the repaired parts is effected by immobilizing the fingers and hand in a moulded plaster of Paris splint. Where there is a history of soil contamination attending the injury, a prophylactic injection of tetanus and gas bacillus antitoxin is given routinely.

Postoperative Treatment. Supervised gentle and purposeful early active movements of the fingers are begun within forty-eight hours after the operation, while the muscles are relaxed in splintage. The movements should be done under the direction of the surgeon, and are carried out by having the patient move both hands simultaneously. Cautious but complete excursions in flexion and extension of the fingers are carried through several phases and repeated twice a day. We find that by coördinated action of both hands in unison, the act, being purposeful, becomes easier. In this we agree with Harmer[6] who advocates early supervised motion following repair of tendons without neural involvement. No force is exerted upon the tendons before the third week, and when instituted, it is done slowly and carefully.

This early motion has a dual effect—it prevents agglutination between tendon and tendon sheaths, obviating the formation of later adhesions, and the tension exerted upon the suture line stimulates the early formation of a callus weld. It also tends to lessen the period of splintage, maintaining muscle tonus, and restoring early function. The average period of splintage in this series of cases was ten days.

Postoperative Infection. The incidence of early postoperative infection has become a relatively rare occurrence, as the result of gentle but thorough cleansing and débridement of the wound before repair is instituted. There are instances, however, where infection and breakdown of the operative wound occurs in spite of every preoperative and operative precaution. This occurred three times after operations within the "golden period," twice in cases of blows of the closed fist against human teeth with laceration of the extensor tendons and compound fractures of the proximal phalanges; one instance followed cleavage of tendon and phalanges from an axe injury. It is noteworthy that all these cases of early postoperative infection were associated with compound fractures.

There are also instances where delayed breakdown may take place weeks or months after a tenorrhaphy with primary healing. This is manifested by redness, tenderness, and the formation of one or multiple blebs along the operative scar. Incision often reveals each bleb as consisting of a pus blister within which the suture material lies freely exposed. When this occurs, it is necessary to remove the entire offending suture before complete closure will take place. There were four cases of delayed breakdown—two at the wrist, one in a digit, and one in the tendo Achillis. At first, we were inclined to blame the suture material alone for these sequelae, but investigation demonstrated that in some individuals in whom the breakdown of the wound took place, a distant pyogenic focus of infection was present elsewhere in the body and the secondary breakdown at the tendon suture line was most likely disseminated by a hematogenous route.

On the other hand, there were cases where the suture material was extruded through the skin without any evidence of infection present. Some of these sutures were sloughed out as late as three years postoperative. The patient would state that a "black speck" could be seen at first beneath the skin at the suture line. This worked its way through the skin and the "threads hung out." This working of the suture material to the surface is more likely to occur in the fingers, where there is a paucity of tissue intervening between the suture line and the skin, and the constant friction of movement in these parts propels the foreign material to the skin surface and out. This happened six times, and each time it followed suture of tendons in the digits.

ANALYSIS OF CASES

This report is based on ninety consecutive unselected cases of tendon injuries, representative of the common sites in the body where accidental division of tendons occurs. It is, therefore, not limited to injuries in tendons of the hand alone.

These cases are from the Department of Traumatic Surgery, of the New York Post-Graduate Hospital and the Reconstruction Hospital (Dr. John J. Moorhead, Director). They represent personal cases and cases from the services of Dr. Henry H. Ritter and Dr. Herbert Bergamini.

In the group of ninety cases of tendon lacerations reported here, seventy-five were of Type 2 (compound) and fifteen were of Type 1 (not compounded).

The distribution, as to parts involved, was as follows:

	No.			
Hands and fingers	78	*Males* 67	Left	30
			Right	37
		Females 11	Left	5
			Right	6
Tendo Achillis	3	*Males* 2	Left	1
			Right	1
		Females 1	Left	0
			Right	1
Quadriceps extensor	6	*Males* 6	Left	2
			Right	4
Patellar tendons	2	*Males* 2	Left	0
			Right	2
Biceps tendons	1	*Males* 1	Left	0
			Right	1

The total number of males who suffered tendon injuries is seventy-eight, females, twelve. Included with this list were eight children.

Age Incidence. The oldest patient in this group was 76 years of age; the youngest, 2 years. The average age was 28 years.

Causes of Lacerations. Lacerations resulting from sharp instruments were as follows:

Glass, Knife, Axe, Metal, Porcelain, etc.	61 cases	Hands and fingers	58
		Tendo Achillis	3

Lacerations resulting from blunt instruments:

Lead pipe, Iron pipe, Door, Auto, Rock, Window ledge, etc.	14 cases	Hands and fingers	14

Lacerations resulting from muscular violence:

15 cases	Patellar	2
	Biceps	1
	Fingers—Extensors	6
	Quadriceps—Extensors	6

Time Interval from Injury to Surgical Intervention. Forty-five patients were operated on within six hours of injury. In the remainder the time interval was as follows:

24 hours	19
3 days	8
1 week	7
2 weeks	4
4 weeks and over	7

Of the eighteen cases in which the delay was one week or more, seven were Type 1 avulsions of the extensor tendons of the fingers from their insertion without break in the skin; five were lacerations of the flexor tendon at the wrist that were overlooked in suturing a seemingly superficial laceration of the skin, and came to secondary operative repair from one to four weeks after injury; three were overlooked flexor tendons in laceration of the palm, operated on within ten days; three were quadriceps extensor tendons.

The end results following tendon lacerations when treatment is carried out systematically under guided supervision often gives a complete restoration of function, and the period within which this is accomplished is measured, not in terms of days or weeks, but rather in that of the intelligence and coöperation of the patient to execute these early movements.

In arriving at the functional end results in tendon injuries, the numerical ratio and mathematical grading adopted by Moorhead[6] for fractures was used: 60 per cent for function, and 20 per cent for union and contour. Function, or the capacity to perform, is measured in terms of the ability fully to flex or extend the part, as the case may be. Union, or the state of repair, is measured by the character and extent of the tendon callus which may be sufficient, exuberant, or attenuated, lengthening the

SITES OF LACERATIONS IN HAND

	No. of Cases	Flexors			Extensors		
		Total	Left	Right	Total	Left	Right
Thumb	9	6	1	5	3	3	0
Index	20	7	5	2	13	3	10
Middle	13	3	1	2	10	4	6
Ring	2	1	1	0	1	1	0
Little	5	0	0	0	5	3	2
Wrist	23	13	6	7	10	5	5
Hand	5	1	0	1	4	3	1
Forearm	1	0	0	0	1	0	1
	78	31	14	17	47	22	25

SITES OF LACERATIONS IN DIGITS*

	Total	Joint	Flexor			Extensor		
			Total	Left	Right	Total	Left	Right
Thumb	9	A	4	1	3	3	3	0
		B	2	0	2	0	0	0
		C	0	0	0	0	0	0
Index	20	A	3	1	2	4	2	2
		B	4	4	0	4	2	2
		C	0	0	0	5	0	5
Middle	13	A	1	0	1	4	1	3
		B	2	1	1	4	1	3
		C	0	0	0	2	0	2
Ring	2	A	1	1	0	1	1	0
		B	0	0	0	0	0	0
		C	0	0	0	0	0	0
Little	5	A	0	0	0	3	1	2
		B	0	0	0	2	2	0
		C	0	0	0	0	0	0
	49	..	17	8	9	32	13	19

Total of Injuries at "A" Joint—Flexor..... 9
Extensor..... 15
Total of Injuries at "B" Joint—Flexor..... 8
Extensor..... 10
Total of Injuries at "C" Joint—Flexor..... 0
Extensor..... 7

* The metacarpal phalangeal, proximal and distal interphalangeal joints are designated here as "A," "B," and "C" joints, respectively.

tendon. Contour is measured in terms of deformity, swelling due to excessive tendon callus, and appearance of operative scar or scars.

End results were obtained in eighteen cases in this series. These included four cases of laceration at the flexor surface of the wrist, in two of which the median nerve was involved; three cases of lacerations of the flexor tendons in the palm; two cases of lacerations of both superficial and deep flexors in the digits, three cases

NUMBER OF TENDONS INVOLVED

No. of Tendons Involved	No. of Cases	Flexor			Extensor		
		Total	Left	Right	Total	Left	Right
1	35	10	3	7	25	10	15
2	21	13	7	6	8	4	4
3	12	1	0	1	11	0	11
4	7	4	1	3	3	1	2
5	2	2	0	2	0	0	0
10	1	1	0	1	0	0	0
	78	31	11	20	47	15	32

Total number of tendons sutured in 90 cases.. 173
Hand, wrist and fingers..... 161
Others..... 12

of lacerations of the long and short extensors of the thumb; two cases of avulsion of the extensor tendons of the fingers from their insertion; and two cases of lacerations of the tendo Achillis. In this group, two were Type 1 and sixteen were Type 2; fifteen were males and three were females.

No case was evaluated for end result earlier than three months, this being regarded sufficient time for complete restoration of tendon function. The average grading for all the follow-up cases was 85 per cent, divided into 50 per cent for function, 17 per cent for union, and 18 per cent for contour.

CONCLUSIONS

1. Division of tendon injuries into Type 1 (simple, without a break in the skin) and Type 2 (compound), places these injuries in the same standardized classification as skeletal injuries, of which they are a complement.

2. This practical classification automatically divides the Type 1 group into the "wait and see" class, and Type 2, into the "look and see" class.

3. Judged by this group of cases, no age is spared. Sharp instruments lead in the causation. No tissue is respected; cutaneous, neural, vascular, and bony tissues may share in the damage by the impact that severs the tendons. Any superficial injury, no matter how seemingly trivial, which reflects a suspicion of deeper struc-

ture involvement, should be investigated surgically for possible damage to underlying tissues.

4. This series of cases confirms the greater relative incidence of tendon injuries in the male, the proportion being about six to one; the greater incidence of Type 2 injuries; the more frequent involvement of the right extremity; the more frequent affection of the dorsal surface; and long extensor of the thumb lead in frequency, in the order given, among the extensor tendons.

6. In freshly compound lacerated tendons, the time of repair (six hours) and thorough debridement are far more important than the character of suture material and the type of repair.

7. Early debridement and repair diminish the incidence of postoperative infection,

TENDONS INVOLVED IN HAND

	Total	Compound				Not Compounded			
		Complete		Incomplete		Complete		Incomplete	
		Left	Right	Left	Right	Left	Right	Left	Right
Flexor palmaris longus	11	4	7	0	0	0	0	0	0
sublimis digitorum	15	8	7	0	0	0	0	0	0
profundis	14	7	7	0	0	0	0	0	0
carpi radialis	6	2	4	0	0	0	0	0	0
carpi ulnaris	3	1	2	0	0	0	0	0	0
pollicis longus	6	2	3	0	1	0	0	0	0
pollicis brevis	2	1	1	0	0	0	0	0	0
	57	25	31	0	1	0	0	0	0
Extensor communis digitorum	16	3	11	0	0	0	2	0	0
pollicis longus	11	5	5	0	1	0	0	0	0
pollicis brevis	5	3	2	0	0	0	0	0	0
pollicis indicis	15	5	3	1	1	1	4	0	0
minimi digiti	11	3	7	1	0	0	0	0	0
	58	19	28	2	2	1	6	0	0

NERVE INJURIES

Total number of cases with nerve involvement	7
Median nerve	5
Ulnar nerve	2

In case both the median and the ulnar nerves were involved.

the wrist and fingers, particularly the index and middle fingers. The level of the "A" joint is the commonest site of tendon severance in the digits. In the lower extremities, the quadriceps extensor tendon, the tendo Achillis, and the patellar tendons lead, in the order mentioned.

5. Of the flexor tendons, the flexor sublimis, profundus digitorum and palmaris longus lead, in the order given, while the extensor communis digitorum, indices

but do not altogether eliminate delayed breakdown of the operative wound or later extrusion of the suture material without breakdown of the wound.

8. Fine black silk is the suture of selection, and the Bunnell stitch is the method of choice.

9. Early preliminary preoperative relaxation by splintage is important in facilitating and obtaining a good repair, while early regulated supervised post-

operative motion of the fingers (forty-eight hours postoperative) is essential in obtaining functional restoration of hand.

10. The index finger, thumb, and middle fingers are, the important digits. One must always try to save the outer side of the hand where the severity of the injury demands sacrificing parts of this impor- that human tool. However, in the foot, one attempts to spare the inner side, this being the more important functional part of that member.

11. Patience is the keynote in treating tendon injuries: patience in evaluating the amount of damage present; patience in repairing the damage wrought; and patience in restoring functionally what one has restored surgically.

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Variations in the degree of pain produced in inflammation depend on variations in the degree and location of the swelling. Thus, soft tissue inflammation is less painful than that developing in some rigid structure such as bone.

VISCERAL COMPLICATIONS IN TUBERCULOSIS OF BONES AND JOINTS*

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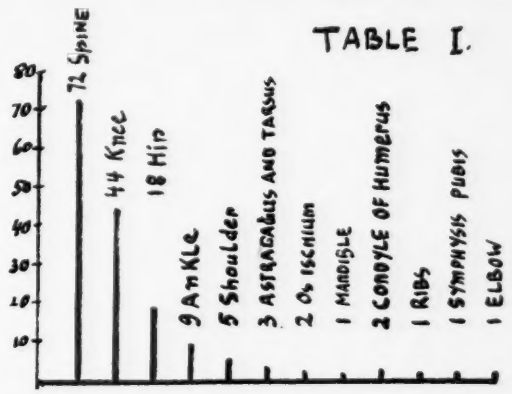
TUBERCULOSIS of the skeletal apparatus remains one of the most difficult problems in surgery, difficult because of the surgical technicalities involved and because of the chronicity of the lesion. Consequent to this chronicity is the ever present threat of visceral complication, a threat which, unfortunately, we are apt to await rather than to anticipate.

The present study is designed to investigate the nature of visceral involvement in tuberculosis of the bones and joints, and to determine to what extent it follows a predictable path. Is it possible to establish any rules enabling us to anticipate complications? At the outset of this investigation it was decided not to include cases presenting a terminal miliary dissemination since its pathway from the blood stream is rather diffuse and the sequence of visceral involvement speculative. Furthermore, it is not the intention of the author to enter into any discussion of theoretical concepts or to restate any of the physiologic or pathologic material easily available in current literature.

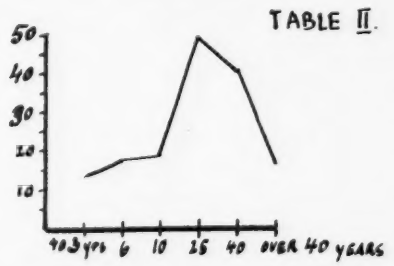
The material from which the present conclusions were derived consists of unselected cases of tuberculosis of bones and joints admitted to the Hospital for Joint Diseases and Mount Sinai Hospital for the five year period 1932–1936 inclusive. Out of a larger series, 159 cases were selected for study, these being all the cases definitely proved as tuberculous and presenting sufficient data to warrant interpretation.

Table I shows the distribution of the lesion in all 159 cases. Table II shows the distribution of the cases as to age, and Tables III and IV the distribution as to sex and age.

These data require no further discussion here since their pertinence to the present subject is incidental. Briefly, seventy-two cases involved the spine, thirty-five male and thirty-seven female; 44 cases involved



the knee, evenly divided between the sexes, and eighteen cases occurred in the hip joints, ten male and eight female.



Of these cases, ten showed evidence of visceral complications:

1. A 59 year old male had tuberculosis of the right hip and developed bilateral tuberculosis of the kidney.

2. A 39 year old male with tuberculosis of the spine developed perinephritic abscess.

3. A 40 year old female with tuberculosis of the spine developed Addison's disease.

* From the Surgical Service, Hospital for Joint Diseases, and the Orthopedic Service, Mount Sinai Hospital, New York City.

4. A 25 year old male with tuberculosis of the spine developed hydronephrosis. No tubercle bacilli found in urine.

5. A 26 year old male with tuberculosis of the spine developed epididymitis.

6. A 5 year old female with tuberculosis of the right knee developed tuberculous peritonitis.

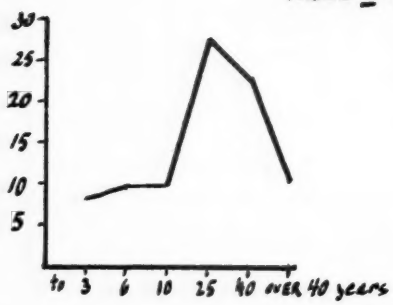
7. A 3 year old male with tuberculosis of the spine developed left renal tuberculosis.

8. A 35 year old male with tuberculosis of the spine developed toxic hepatitis.

9. A 2 year old male with tuberculosis of the right mandibula developed left renal tuberculosis.

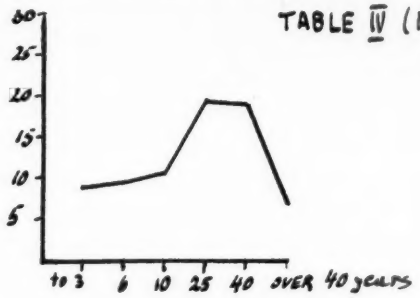
10. A 20 year old female with tuberculosis of the spine developed left renal tuberculosis.

TABLE III (FEMALE).



Looking over these cases it immediately becomes apparent that most of the complications were secondary tuberculous infections, or possibly foci present without symptoms before the onset of perceptible bone and joint manifestations. Whichever it was, following or antedating the skeletal manifestations, insofar as the existing status was concerned, they presented a complication of the presenting lesion. Predominantly the complicating infection involved the genitourinary system (eight cases, 5 per cent). There were four cases (2.5 per cent) of proved renal tuberculosis and one case of proved epididymitis tuberculosa. These figures are higher than the findings of Duncan who reported genitourinary complications in 2.5 per cent. Snyder reported a still higher percentage. Of his 100 cases, eight were found to have renal tuberculosis and five tuberculous epididymitis. Harris, who systematically examined the urine of forty-three adults and sixty-seven children for tuberculous bacilluria, found tubercle bacilli in the urine in sixteen cases of the former and nine of the latter. Of his sixteen cases he reported five as certain tuberculosis of the kidney (11.6 per cent) and six as uncertain. He had no case of proved tuberculosis of the kidney in his juvenile cases.

TABLE IV (MALE).



In the present series, the hydronephrosis of Case IV was caused by pressure of a cold abscess on the ureter. The kidney was shown not to be involved by tuberculosis. The occurrence of adrenal involvement in Case III with the symptoms of Addison's disease is exceptionally interesting because of its rarity in the presence of other easily recognized gross tuberculous lesions. As for the hepatitis in Case VIII, it is hard to say whether it was of tuberculous origin or not. The hepatitis cleared up completely after a few weeks.

The repeated demonstration of secondary genitourinary involvement in skeletal tuberculosis should suggest the advisability of examining the urine of all patients suffering from tuberculosis of bone and joints at regular intervals for tubercle bacilli, so that lesions might be discovered early. Since early renal tuberculosis gives no lead to the examiner because of lack of symptoms, repeated examination of the urine should be done as a routine. The observation that tubercle bacilli may appear intermittently minimizes the significance of negative findings. Many progressive cases of kidney tuberculosis or tuberculosis of the ureter and bladder

could be thereby anticipated and proper measures instituted.

SUMMARY

1. One hundred fifty-nine cases of proved tuberculosis of bones and joints were examined, ten of which showed visceral complications.

2. Four cases of renal tuberculosis were found and one case of epididymitis.

3. All cases except one have secondary tuberculous lesions as complications.

4. The most frequent is the involvement of the genitourinary tract.

5. Routine examination of the urine for tubercle bacilli at repeated definite intervals should be included in the general supervision of cases of tuberculosis of the skeletal system.

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The inflammatory reaction . . . is just as physiological as the healing process, since it is a response of nature which prepares the ground for the repair of lost, divided or dead tissue.

A NEW CLOSED METHOD OF TREATING SUPRACONDYLAR FRACTURES OF THE ELBOW

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SUPRACONDYLAR fractures in infants and children are very common and generally present a distinct therapeutic problem. They are usually displaced and oftentimes comminuted with a great amount of accompanying soft tissue trauma. A careful thorough search should be made for any neural disturbance before reduction is attempted. Reduction should be done with the least amount of force, as gentleness in the entire manipulative procedure is of the utmost importance. Usually the acute flexion position can be counted upon to maintain the corrected osseous alignment, but this flexion is a very potent potential cause for Volkmann's ischemic paralysis. A too tightly applied bandage may within a few hours lead to unpleasant sequelae, but if it is loosened there is the likelihood of letting the fragments become somewhat displaced again.

Anterior and posterior moulded plaster of Paris splints and many other types of rigid immobilizing devices are being used and often are excellent. However, these appliances demand the elbow to be at not too acute flexion, and hence, at the start are at a disadvantage as a means of retaining the reduction. The pull of the triceps is ever present and must be counterbalanced by some mechanical method. Recently good results have been reported by the use of gravity pull with the wrist held by a sling and the elbow slung by this means in moderately acute flexion.

There are several surgical procedures for the treatment of this fracture, the simplest being the use of the Kirschner wire through the olecranon process with or without a plaster of Paris cast. There are many fractures about the elbow, which have to be operated upon with screw, nail or wire internal fixation. There are also other cases which do very well with the use of tongs. Several other operative procedures are being used in the treatment of this injury.

The method, the description of which follows is, I believe, original, as I have not seen any mention of it in the literature. This procedure does not claim to be a panacea for supracondylar fractures, but is only to be used where it is applicable. However, in many cases it can be tried and if it does not prove successful other more drastic methods can still be undertaken. It has now been used on three patients with very good success; a short record of these is found below.

I have employed vinethene as an anesthetic in many dozens of infants with fractures and dislocations, and I find it excellent for the average case. A procedure lasting thirty minutes or thereabouts can usually be done very well under this anesthetic, ordinarily with good relaxation. In certain cases ether has to be given as a supplement but only for the terminal part of the procedure. Vinethene is especially useful in an office or clinic practice, and is being used a great deal in adults as a preliminary anesthetic. There is very little post-anesthetic nausea, vomiting or excitement, and the toxicity of the drug is very low. In this respect it markedly differs from ethyl chloride, which has to be used with great caution and for only a short time. In older children novocaine as a local anesthetic can be used, but usually this type of anesthetic is not suited for youngsters or infants.

The various steps in the application of the retention cast are shown in the accompanying photographs. The fracture is usually better reduced before the plaster of Paris cast is applied, although in one

instance I found it more advantageous to put on the plaster cuff first. After reduction the metal stirrup is incorporated in the plaster cast. Which order to follow will depend on the individual case and whether the plaster cast will in any way interfere with the manipulation of the fractured fragments.

Fig. 1. Stockinet and anterior felt pad applied, the latter ½ to ¾ inch thick. This is usually sufficient to prevent untoward pressure symptoms. (The adhesive strips are only to hold the felt in place for the photograph.)

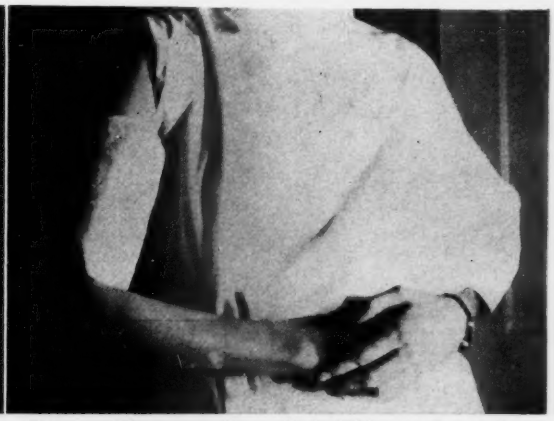

Fig. 2. Plaster cuff applied. This need not be thick or bulky.

Figure 1 shows the stockinet and anterior felt pad applied, the latter being ½ to ¾ inch in thickness, which usually is sufficient to prevent untoward pressure symptoms. (The adhesive strips are only to hold the felt in place for the photograph.) Figure 2 shows the plaster cuff applied; this need not be very thick or bulky. Figure 3 shows the duralumin metal U splint, which is shown incorporated in the plaster cast. In my three cases I used a duralumin Böhler walking stirrup with the rubber walking tread removed and the side arms cut down to the desired length. The bottom of the bar should be about two fingerbreadths below the olecranon process. Figures 4, 5 and 6 show different views of the leather belt, which is looped around the arms of the splint. Between the anterior loop of the belt and the supracondylar aspect of the elbow is placed a thick piece of the felt. This is a necessary precaution in order to prevent too much pressure on the underlying structures. The belt can be tightened up until enough pressure is obtained to keep the supracondylar fragments in alignment with the proximal shaft fragment. With the arms of the metal splint placed slightly anterior to the line of the condyles enough space is allowed for the desired pressure to be applied by the belt. If the stirrup has been applied too far posteriorly and the belt flattens out too much when pulled tight, added felt strips may be applied which will put the pressure surface further posteriorly. This will increase the effective pull or pressure of the belt. Either fluoroscopic or *x*-ray control combined with clinical check-up will be necessary to determine when sufficient reduction and immobilization has been obtained.

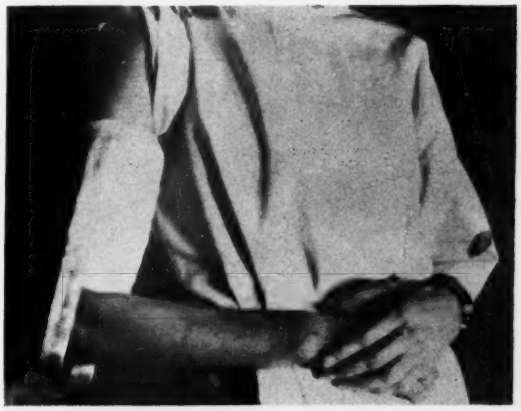

Fig. 3. Duralumin metal U splint incorporated in plaster cast.

The extremity is best carried in a sling with the elbow at a right angle, although

more or less flexion may be obtained if desired. In one case I used just a sling around the wrist, which embodies the principle of the gravity pull method. The movement of the forearm at the elbow has no effect whatever on the fractured part if adequate pressure is obtained by means of the apparatus. The triceps pull will naturally tend to displace the distal fragments posteriorly and upward, but this can be lessened by having the elbow in the mid position between right angle flexion and extension.

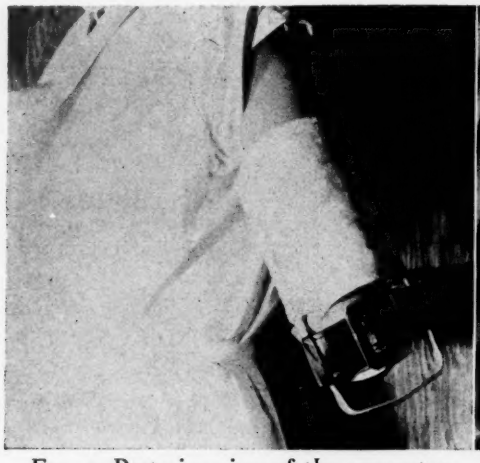

Fig. 4. Posterior view of the apparatus with the leather belt in place.

Fig. 5. Lateral view of the apparatus.

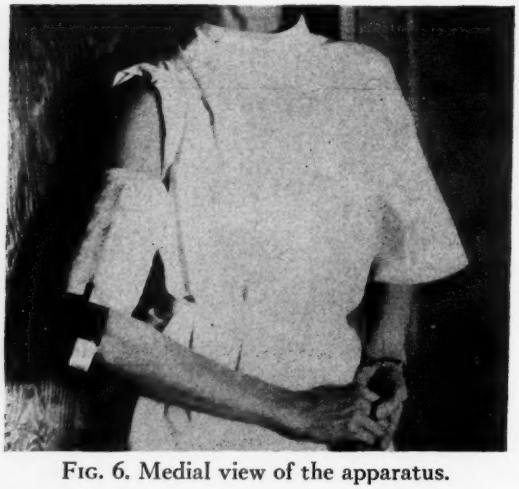

Fig. 6. Medial view of the apparatus.

A careful watch must be maintained for signs of circulatory or neural involvement, and the extremity should be elevated on one or two pillows for a few days after reduction, during which time the patient had better be kept in bed. If any dangerous swelling appears or other undesirable signs or symptoms are seen the belt should be loosened and the elbow extended somewhat. I have found that the belt can be tightened or loosened during the first few days after reduction without the patient's experiencing much of any pain.

The follow-up on these cases entails nothing unusual. The apparatus gives excellent protection to the involved region from falls or blows by playmates and others. I have noticed much less complaint from such minor traumata from these three patients so treated than from the ones I have had up in Jones or Ashhurst bandages or in moulded plaster of Paris splints.

CASE HISTORIES

Case 1. A 12 year old boy sustained a supracondylar fracture of the right humerus in a fall from a bicycle. Figures 7 and 8 show the anterior-posterior and lateral views of the injury. The fracture was reduced one hour or so after injury under general anesthesia and put up in the Jones position. The following day the bandage had to be loosened somewhat due to circulatory impairment from swelling about the elbow. *X*-rays taken at this time showed some posterior and upward displacement of the lower fragments. The patient was re-anesthetized and the fracture reduced and held in alignment by the author's method. The cast and splint were removed in four and one-half weeks, during which time there had been no circulatory involvement. A partial radial palsy was noticed shortly after the injury and has gradually

improved. Figures 9 and 10 show the condition of the involved member about three months later. The motions of the elbow at this time

Fig. 7. Case 1. Anteroposterior view of the elbow before reduction.

Fig. 8. Case 1. Lateral view before reduction.

were from 110 to 70 degrees. The anterior-posterior view shows more of a varus condition than was clinically discernible. When last seen, five months after the accident, the patient had a moderate degree of varus deformity. The motions were from 155 to 55 degrees, and he was active in all manner of sports. He had still some radial nerve involvement, which was continuing to improve slowly.

This patient's nerve injury was thought to be due to a direct trauma at the time of accident and was not caused by any ischemic complication. At no time was the swelling of the involved part great enough to produce any numbness, blueness, edema or coldness of the fingers, whereas the original *x*-ray clearly shows the great amount of displacement and jagged edges of the fragments.

Case II. A 13 year old girl fell from a limb of a tree eight feet from the ground, landing on her right upper extremity. She sustained a Colles fracture of the right wrist and a supracondylar fracture of the right elbow, involving the lateral condyle with some posterior and outward displacement.

The patient was anesthetized about two hours after the accident, and the wrist reduced and held by anterior and posterior moulded plaster of Paris splints from the fingers to the elbow in the Cotton-Loder position of palmar flexion and some ulnar deviation. The elbow fracture was reduced, and another pair of 3 inch anterior and posterior moulded plaster of Paris splints were placed on the limb from the shoulder to the fingers, overlying the previously applied forearm ones. The elbow was placed at about 45 degrees flexion. Then a circular plaster cast with the incorporated duralumin walking stirrup was applied to the arm, according to the author's method. The pressure belt was placed directly on the posterior plaster splint which was well padded over the lower humerus. Post-reduction *x*-rays were taken through the plaster splints of the wrist and the elbow and

revealed excellent position of the fragments. This patient was watched very carefully for twenty-four hours. The elbow had to be let down from 45 degrees to 60 degrees during the night. However, no serious complaints developed. The entire immobilizing device was removed from the extremity exactly one month later and *x*-rays showed good position and firm bony union of the involved bones. The patient has done very well with complete return of motion and power in the wrist, but some limitation of motion in the elbow. She is left handed and it is a great task to get her to use the right hand. Motions of the elbow are from 140 degrees to complete flexion and she is getting along splendidly.

FIG. 9. Case I. Anteroposterior view three months post trauma.

FIG. 10. Lateral view of same case three months post trauma.

CASE III. A 6 year old boy was seen here for a malunited supracondylar fracture of the left humerus. The patient had sustained the injury about four weeks before entry. Open correction with osteotomy was done and the elbow immobilized by author's plaster cuff-metal stirrup method. The apparatus was removed four weeks postoperatively. The lateral view showed some posterior displacement of the lower fragment but new bony formation on the posterior aspect of the humerus is tending to correct this misalignment. When last seen, ten weeks postoperatively, he had painless free motion from 80 degrees to 120 degrees and was rapidly using the extremity for almost everything. This child is one of our State crippled children cases. Later on another osteotomy to get a better alignment of the lower fragment may be done. In this case, however, the cuff-stirrup retentive apparatus held the distal fragment satisfactorily without any neurological or circulatory disturbance.

SUMMARY

A new method of retention of the reduced supracondylar fracture is described, which has the following advantages: (1) It is a closed method. (2) It is easy and simple to apply. (3) It adequately holds the elbow in place and the pressure on the distal fragment can be easily increased or decreased as desired. (4) It allows free motion of the elbow. (5) There is no constricting device in the antecubital region.

SUBMUCOUS HEMORRHOIDECTOMY

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NEW YORK CITY

HEMORRHOIDECTOMY is one of the most universally performed operations in the realm of the surgical specialties. An important predisposing factor in the etiology of hemorrhoids in women is the increase in intravenous pressure by the gravid uterus. Prolonged labor and difficult delivery aggravate this condition. In my gynecologic practice I had often been requested by patients to remove existing hemorrhoids while repairing lacerations of the pelvic floor. This I refused to do for fear of unduly prolonging the time of the operation, and because of my reluctance to expose the patient to the additional suffering which accompanies the usual hemorrhoid operations.

Because of these objections, I searched for an operative procedure which would meet the following conditions:

1. The method should restore the anal and lower rectal region to as near normal as possible, with no complicating postoperative stricture.

2. The operation should be as free from postoperative pain and hemorrhage as possible.

3. It should be simple of execution so that it could be performed with the least possible trauma and in the shortest possible time.

I feel that I have devised a modification of the ligature and excision technique, a simplified submucous hemorrhoidectomy, which fulfills these requirements. In order that one may be able to evaluate the advantages that this method has over the older operations, a fair acquaintance with the pathogenesis of hemorrhoids is necessary.

From the various descriptions of the pathology of hemorrhoids by Brav,[1] Fleischer,[2] Montague,[3] and Buie,[4] the following brief résumé is sufficient to acquaint one with the subject. The hemorrhoidal plexus of veins is the primary seat of this disease and the rectal mucosa is only secondarily involved. The pathologic changes begin with the formation of varicosities in the veins. The increase of the local intravenous pressure arising from constipation, excessive use of laxatives, straining at stool, pregnancy and tumors, produces venous congestion with marked tortuosity of the veins. The walls of these vessels become thinned out, stretched and sacculated. The mucosa overlying such veins becomes attenuated and is loosened from its submucous attachment. The injured vessel with its surrounding areolar tissue and the redundant mucosa form a sacculated mass which on further stretching may eventually become pendulous and protrude through the anal orifice forming what is called a prolapsed hemorrhoid. These varicosities encroach on the lumen of the lower bowel so that the passage of hard fecal masses traumatizes the tissues, causing abrasions and ulcerations of the thinned out mucosa. This process may affect the blood vessels and lead to bleeding, a common symptom of hemorrhoids. Such open wounds, present in an area where pathogenic organisms abound, may easily become infected, resulting in round cell infiltration and chronic inflammatory changes of the vessel walls. Occasionally thrombosis of the veins takes place; the clots may be completely absorbed or they may break through the vessel wall and be extruded. In other instances sclerotic perivascular changes occur in the areolar tissue supporting the veins, thus bringing about the complete occlusion of the vessels. These same pathologic changes take place where external hemorrhoids are present

except that instead of the mucosa being involved it is the skin that covers the varicosed or thrombotic veins.

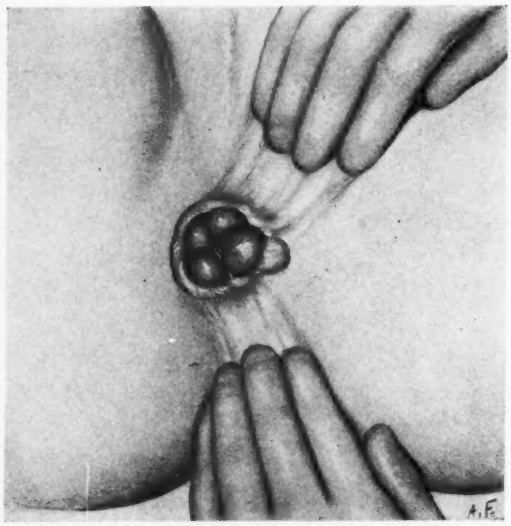

Fig. 1. Assistant bringing operative field into view by manual retraction thereby exposing the hemorrhoid in the process of excision.

TECHNIQUE

The method which I employ is based on a sound conception of the pathologic changes encountered in the lower bowel. None or very little of the rectal mucosa is sacrificed; only the diseased veins with the supporting perivascular areolar tissue are removed.

As the patient is being operated upon for some defect in the genital structures about the pelvic floor she is already under some form of anesthesia and in the usual lithotomy position. Otherwise, local anesthesia of 1 per cent novocaine injected around the anal ring will render the operation completely painless.

In dealing with internal hemorrhoids alone, I proceed as follows: No preliminary dilatation of the sphincter ani is undertaken. This is definitely contraindicated, for it causes traumatism to the sphincteric tissues. It is unnecessary, since the sufficient exposure of the diseased area is readily accomplished by the more gentle manipulation further described.

With the assistant bringing the operative field in view by proper manual retraction (Fig. 1), a small transverse incision is made at the mucocutaneous junction of the exposed hemorrhoid. The free edge of the incised mucous membrane is grasped gently with tissue forceps or Allis clamp, and with a small blunt curved flat-blade scissors the mucous lining is undermined along the entire length and width of the underlying hemorrhoidal tissue. (Fig. 2.) This is followed by a longitudinal incision with a blunt straight scissors along the midline of the mobilized flap, thus exposing the hemorrhoid in its entirety. (Fig. 3.) A No. 00 plain catgut suture on a small curved round needle is passed around the distal end of the hemorrhoid and ligated. (Fig. 4.) This suture will not be absorbed before two or three days, which is long enough to prevent oozing and render the operation almost bloodless. A small hemo-

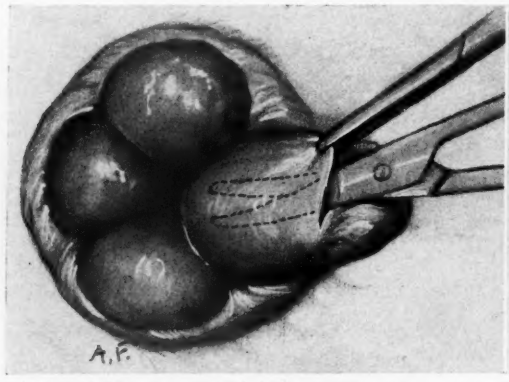

Fig. 2. Transverse incision at mucocutaneous junction and mobilization of rectal mucosa covering hemorrhoidal mass.

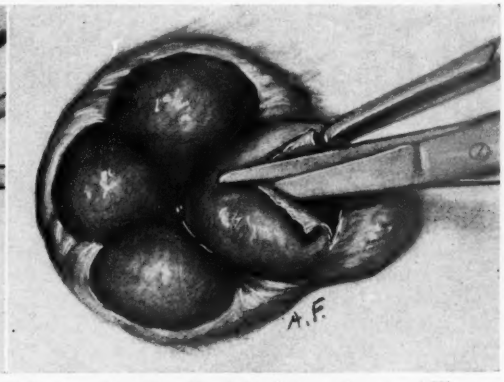

Fig. 3. Longitudinal incision along midline of mucosal flap exposing underlying hemorrhoid.

stat is placed in front of the suture-ligature and the hemorrhoid is removed with either knife or scissors from its submucous bed, care being taken to cut in front of the hemostat and not beyond the ligature. (Fig. 5.)

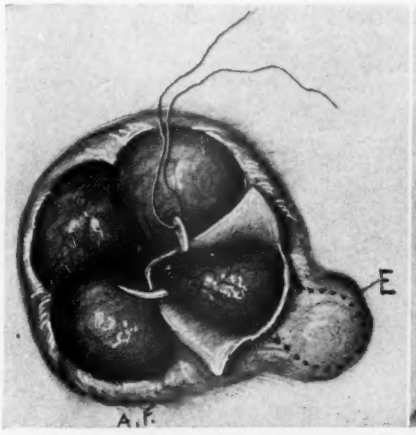

Fig. 4. Suture-ligature passed around distal end of hemorrhoid. E, outline of portion of external hemorrhoid to be removed.

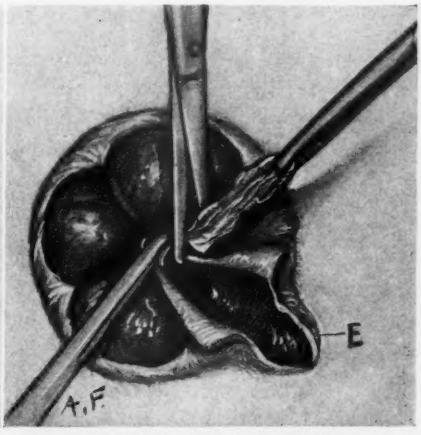

Fig. 5. Excision of internal hemorrhoid. E, external hemorrhoid removed.

The operative field is now covered with two flaps of mucosa. (Fig. 6.) Should these be too redundant, they may be easily trimmed to the desired width, always remembering that it is better to leave behind an excess of tissue rather than too little. If the rectal mucosa is ulcerated, as when dealing with ulcerated hemorrhoids, the diseased portion must be trimmed off in order to insure proper union of the flaps. These are not sutured together but are left free to agglutinate to the underlying raw surface. This process takes place very soon, usually within the first three or four days after the operation.

In a number of mild cases of hemorrhoids I omitted the suture-ligature around the base of the hemorrhoid and, instead, allowed the hemostat to remain in place while removing the hemorrhoid in the manner above described. No bleeding took place after the removal of the instrument. The clamping of the veins for a short time sufficed to prevent bleeding. This adds another important feature in favor of this operation, since ligatures act as irritants, and sutures cause constriction and breaking down of tissues.

When external hemorrhoids are also present, I proceed in a similar manner by mobilizing the skin covering the external hemorrhoid before removing the mass. A sufficient v-shaped portion of the redundant skin is excised, care being taken not to remove too much. This prevents the formation of an ectropion. (Fig. 5E.) The

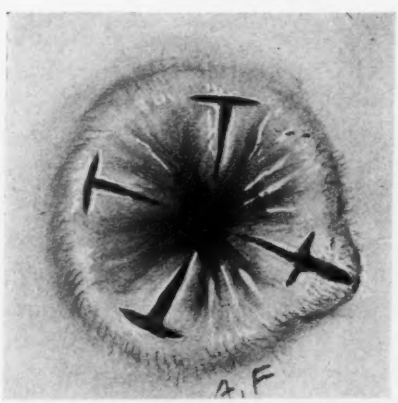

Fig. 6. Operative field after hemorrhoidectomy is concluded.

edges of the skin flaps, like those of the mucosa, are not sutured together. They are left free to agglutinate and usually heal within seven to eight days.

As a rule there is no bleeding from the internal hemorrhoid wound, although there may be just a slight ooze which soon ceases spontaneously. Should there be more than

the usual amount of bleeding from the external hemorrhoid wound, a dry gauze pad held tightly by adhesive strips will stop the oozing effectively. No elaborate perineal dressing is required, just a sterile gauze pad, to be changed when soiled.

Rarely do patients complain of any marked postoperative pain. Should this occur, a hot water bag over the perineum will give quick relief, and in extremely nervous patients a hypo of morphine may be resorted to.

The older methods of hemorrhoidectomy commonly used, the ligature-with-excision and the clamp-and-cautery operations, do not meet with the conditions stipulated above. The clamping off of a large portion of the rectal mucosa with the underlying hemorrhoid, followed by excision of the mass with or without cauterization, leaves behind a large raw surface. Healing takes place slowly by granulation and scar formation. If several hemorrhoids are removed at the same time, the resulting scars may be so extensive as to constrict the lumen of the bowel and form a rectal stricture, a complication noted by Tice.[5]

Severe postoperative pain is usually present for a long time on account of the reaction following the constriction, traumatization and burning of the tissues by the use of the ligature and the clamp and cautery.

Frequent primary postoperative hemorrhages occur because of the wide granulating surfaces remaining after these operative procedures. Such open exposure may lead to bacterial invasion and severe secondary hemorrhage, as noted by Pennington[6] and Barnes[7] and also by Gabriel[8] in his review of a series of 438 hemorrhoidectomies.

The after-care of these cases is governed by the type of operation originally performed on these patients. Where no restriction exists, I use the following routine: I do not constipate my patients for any length of time after the operation. I prescribe a tablespoon of mineral oil to be taken morning and evening for several days beginning with the first postoperative morning. The patients are allowed the use of the bedpan or toilet as soon as the desire for defecation arises. Many patients move their bowels on the second postoperative day with hardly any pain or bleeding.

As to diet, I prescribe liquids for the first day only, followed by a soft diet until the initial movement, after which a regular diet is resumed. It is with great difficulty that I can keep patients in the hospital for longer than three days, for they feel too well to be confined to bed. As a rule I allow them out of bed on the second postoperative day.

There have been no complications in the present series of more than 100 cases. So far, no postoperative hemorrhage requiring special attention was noted. Should this occur, the bleeding point can readily be located and a suture-ligature applied.

Not a single case of stricture of the rectum has occurred up to the present. This can be explained by the fact that the raw surface remaining after the excision of the hemorrhoidal mass is not allowed to granulate and form scar tissue. The two flaps of healthy mucosa or mucosa and skin covering the raw surface agglutinate promptly. The edges unite rapidly and leave only a very fine longitudinal scar hardly visible or palpable six months after the operation.

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RENAL HYDATIDOSIS*

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RENAL hydatidosis, a disease produced by *Taenia echinococcus*, has a low incidence, is not benign, and has been known for the past thirty years through publications. The history of its development falls into three periods: (1) the clinical period; (2) the operative period; (3) the period of pyelography.

ETIOLOGY AND PATHOGENESIS

1. *Circumstances Determining Location.* (*a*) *Frequency.* Nicaise and other authors have reported percentages below the normal 3 per cent. The countries most affected are Algeria, Iceland, New Zealand, Dalmatia, and above all Uruguay and Argentina in South America. My own contribution is represented by twenty-nine personal cases, which form the largest group in medical literature thus far.

(*b*) *Path Traversed by the Parasite.* The arterial route is the most logical and probably the only one.

(*c*) *Primary location.* The cyst is located primarily in the kidney, since in general there is no concomitant lesion in other viscera. Secondary location is exceptional.

(*d*) *Single Cysts.* In general, just one cyst is present, but in exceptional cases the cyst may be multiple or affect both kidneys.

2. *Topography.* The entity hydatid cyst plus the pericystium and its parent membrane have important relationships which I have been the first to stress.

(*a*) *Relationship to the Renal Structure.* Since the cyst originates in the territory of the cortical zone and never in the pyramidal region, it reproduces the forms of the visceral cyst of the liver, destroying the elements of the kidney by the two processes of distention and congestion plus toxemia. Its favorite site is the polar regions and it invades them up to the renal periphery and to the pyelic cavities, where it opens.

Indeed, once the cyst appears in the renal periphery, it will be possible to note that the claim of all authors who have wished to consider the cyst as attached to the kidney (from which it could easily be distinguished) is not justified, since it has been possible to ascertain the following facts:

1. It is impossible to find a line of cleavage which determines where the kidney ends and the cyst begins.

2. There exists a parenchymatous zone which invests and accompanies the pericystium for its entire length.

3. This pericystium is at the same time invested by the renal capsule, as proved by two things: in the first place, the cyst is always intrarenal; in the second place, the cyst is always infracapsular.

Although other authors consider that the cyst is not invested by the renal parenchyma (Fig. 1), my own view is that the cyst is invested by the renal parenchyma and also by the renal capsule. (Fig. 2.)

(*b*) *Relationship to the Pyelic Cavities.* Contrary to current reports, the cyst never opens directly into the pelvis of the kidney without having its base always resting *upon the end of a primary calyx* through a solitary and small hole of communication. As a result of this, we note a special aspect of the cyst: the aspect which will give rise to the pyelographic indication which I have called the "goblet sign." This sign consists in a large oval cavity represented by the pericystium, with one base

* Read before the Urological Section, N. Y. Academy of Medicine, Feb. 16, 1938.

of support upon the extremity of the primary calyx, and with one support represented by this primary calyx.

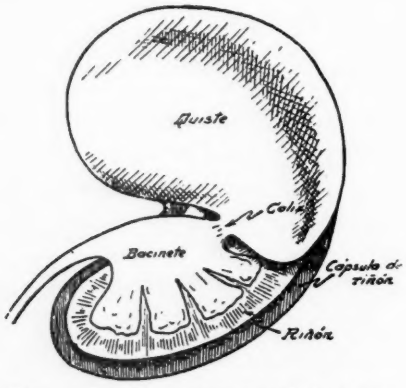

FIG. 1. Classic anatomopathologic picture of a renal hydatid dist. The cyst is covered neither by the kidney nor by the capsule.

The goblet sign may be identified as follows: the pericystium rests upon the extremity of one calyx, which serves as

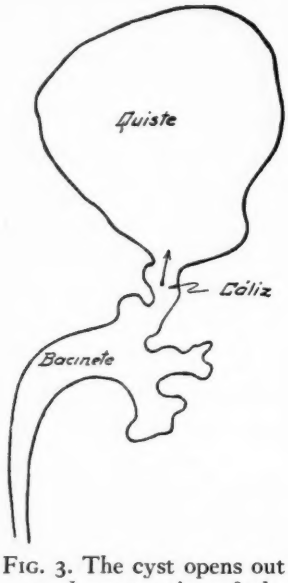

FIG. 3. The cyst opens out at the extremity of the primary calyx, giving the goblet sign.

support to it and assumes the shape of a goblet. (Fig. 3.)

PARENCHYMAL LESIONS

These lesions are important, they can be traced around or far from the cyst, they occur early, and they involve the parenchyma *in toto*, a fact which explains the functional alteration of the organ.

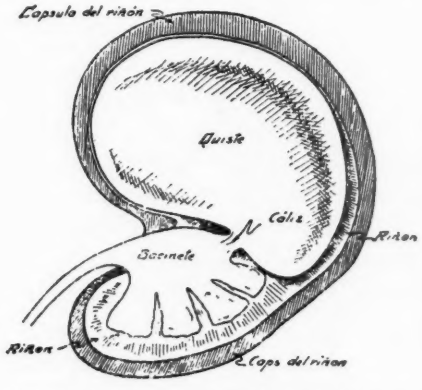

FIG. 2. Our anatomopathologic picture of the same condition. The cyst is always intrarenal and infracapsular.

ANATOMOPATHOLOGIC DEVELOPMENT OF THE CYSTIC SACS

The cystic sacs are frequently regarded as similar to hydropyonephrosis. This, however, is a mistake, for two reasons: (1) the sacs in hydropyonephrosis represent permanent lesions which have no tendency to regress once they have ruptured; (2) on the other hand, once the sacs of a hydatid cyst have burst, they tend to recede, to shrink, and to disappear by fibrous transformation. It is for this reason that I wish to stress the fact that it is not rare to see enormous cysts which had occupied the entire abdomen transformed into small stumps as soon as they have been drained.

SYMPTOMATOLOGY

Renal hydatidosis presents the characteristics of hydatid diseases: slow, progressive, and insidious development; destructive development; and tendency toward infection. Its symptomatology is characterized by tumefaction; functional urinary changes, and general functional changes.

The tumor itself is globular, rounded, *not pitting*, sometimes fluctuating and sometimes like parchment in consistency,

and on rare occasions gives the sensation of a thrill.

When the symptoms are thoracic and abdominal, the hypochondrium is apt to be involved. When the symptoms are abdominal, the flank is apt to be involved.

Fig. 4. A calcified closed cyst, showing the goblet sign.

A cyst of the spleen should not be confused with a renal cyst because the former never arrives inside the angle of the colon. We should further point out that the splenic cyst usually displaces the angle of the colon which never occurs with a renal cyst.

The characteristic feature of renal cystic tumefactions is that the kidney generally loses motility, because the renal cyst, contrary to present claims, soon adheres to the lumbar fossa.

The urinary functional changes are always present. When we are dealing with a closed cyst they are evidenced by microscopic hematuria and functional alteration of the kidney. When we are dealing with an open cyst the above symptoms are exaggerated and there appears the classic triad of hydaturia, nephritic colic, and accordion-shaped tumor.

DIAGNOSIS

We are faced with three possibilities in differentiation: (1) a renal tumor; (2) a cystic tumor; (3) a closed or open cyst. I shall stress here only the two main syndromes: the biologic and the pyelographic.

Biologic Syndrome. Urticaria is very rare. Laboratory tests reveal the following: Eosinophilia ranges between 3 per cent and 10 per cent and can reach 20 per cent in rare cases. However, this point has no absolute value, since it is not evidenced when the cyst is dead and calcified, and may also occur in other diseases. I have found it in 30 per cent of my cases of renal tumors. Cassoni's anaphylactic reaction is found in 60 per cent of the cases, but it may also be present in renal cancer and in neoplasms of the lung. Weinberg's reaction is found in 40 per cent of the cases.

Radiographic and Pyelographic Syndrome. Simple radiography may be of diagnostic aid in two circumstances: (1) in the presence of calcified cysts which are shaped like a goblet or pear and appear in the renal area; this sign is of great value. (Fig. 4.) (2) When there are spots in the kidney region; their value is purely one of presumption.

Fig. 5.

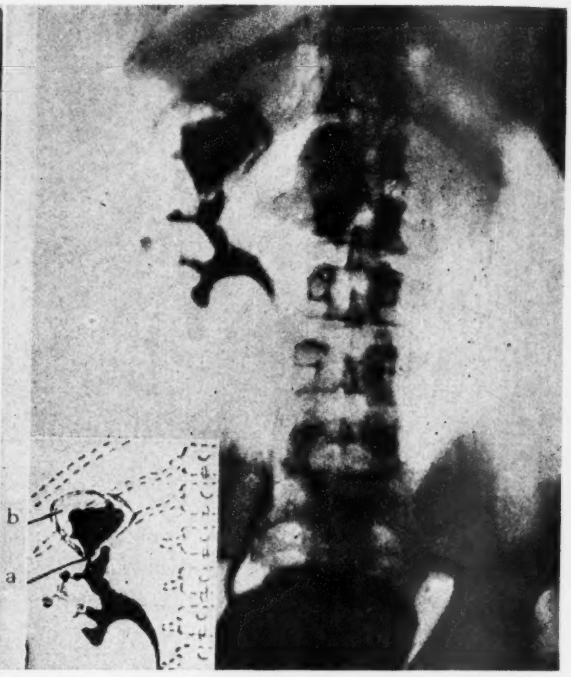

Fig. 6.

Figs. 5 and 6. Open hydatid cyst, showing the goblet sign. (Note the rounded shadows on the inside of the goblet, which correspond to the daughter vesicles.)

Fig. 7. Open hydatid cyst and goblet sign.

Fig. 8. Open renal cyst with pyelogram showing crescent sign.

Pyelography settles the clinical diagnosis and gives picture of the cyst in two eventualities. We are sure of the diagnosis when

we find: (a) what I call the "goblet sign," a broad image resting upon a calyx and taking the form of that calyx. (Fig. 5.)

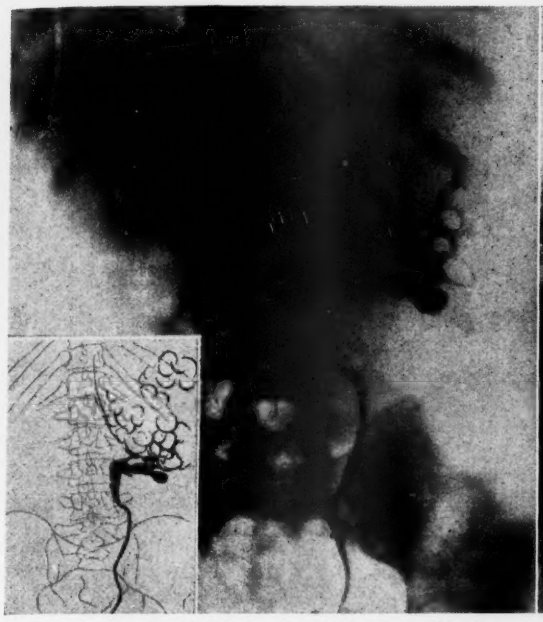

Fig. 9. Open renal cyst with pyelogram showing cluster of grapes sign (honeycomb sign).

Fig. 10. Renal cyst with pyelogram showing horizontal level sign.

The center of the image may be distinct and uniform, or may present a succession of light and dark zones showing rounded regions corresponding to daughter cysts. (Figs. 6 and 7.) (b) *The sign of the crescent* or segment of a moon is of enormous value, and must be distinguished from false crescents (constituted by curvatures of the ureter). (Fig. 8.) (c) *The sign of the bunch of grapes or honeycomb*, the image given by the daughter cysts. (Fig. 9.) (d) *The horizontal level sign* indicates an air cyst of the kidney. This is the only case known to medicine. (Fig. 10.)

These pyelographic data are of fundamental importance, and when the findings present the appearance noted they are pathognomonic of the disease.

The diagnosis may be questioned in certain circumstances: (a) In the case of a ruptured cyst: when there are deformities limited to one calix and simulating crescents; when the rounded appearance of the cyst is outlined; when the areolar aspect is seen to be confined to one calyx. Apart from this, I do not believe that important consideration may be given to amputations of the calyces and displaced ureters mentioned in the literature.

In a closed cyst: where a shadow coincides with the cupola of a calyx; where displacements caused by torsion of the kidney make it appear that the kidney is supporting something, the existence of a cyst may be suspected.

PROGNOSIS

The above mentioned views regarding pathologic anatomy, which show the occurrence of renal destruction, force me to contradict the present views. I believe that a hydatid cyst means an affection endangering the kidney.

TREATMENT

Early treatment and conservative surgery are indicated for hydatid cysts. They must be treated at an early date because their development is progressive and destructive. Surgical treatment must be conservative because they remain a polar affection for a long time, and, above all, because, as I pointed out before, cystic sacs tend to regress and to shrink once

they have burst, being transformed into fibrous stumps.

Nephrectomy is radical treatment. Conservative treatment is constituted by partial nephrectomy, excision of cysts and closure without drainage, and marsupialisation. In practice, we must decide between nephrectomy and marsupialisation.

Nephrectomy is necessary when marsupialisation is contraindicated; it is obligatory in complete destruction of the kidney; in multiplicity of cysts in the same kidney; in calcified fatty cyst, even when the kidney has not suffered much destruction; and when conservative surgery has not succeeded.

In nephrectomy we must not forget that the cyst is always intrarenal and infracapsular, as I have pointed out, and therefore the operation will have to be performed by the infracapsular route (which is easy, possible, and not dangerous). On the contrary the operation is very difficult and very dangerous when, for any reason, it is done by the extracapsular route.

Marsupialization must be borne in mind in all cases; it is also necessary: (1) as a preliminary to total nephrectomy; (2) when required by the patient's condition. It has been unjustly criticized because of the long duration of the fistula. However, I have always seen the fistula disappear within three months whenever the cystic sac was not attached to the wall.

CONCLUSIONS

Renal hydatidosis is a disease whose development is intrarenal and infracapsular, whose diagnosis is determined by pyelographic signs (goblet sign, bunch of grapes sign, crescent sign, and horizontal level sign), whose prognosis is bad, and which it is not always easy to treat.

THE INJECTION THERAPY OF HYDROCELE AND SPERMATOCELE*

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IN medicine as in industry, simplicity, conservation, and economy represent the acme of attainment in any given procedure. To this end, injection therapy for varicosities, herniae and pathologic processes in endothelial-lined sacs has enjoyed widespread application in recent years. This brief exposition will concern itself with a new pharmacologic agent for injection treatment of hydrocele of the tunica vaginalis and spermatocele.

Medical healers from time immemorial have wrestled with the problem of hydrocele. Celsus, in ancient Rome, essayed eradication of the sac by injecting saltpeter and by surgical excision of a portion of the sac. Various surgical procedures from exteriorization to total enucleation of the cystic mass were subsequently tried with varying results. Down the ages, various sclerosing solutions have been used, a partial list including tincture of iodine, phenol, sodium chloride, quinine urea hydrochloride, sodium morrhuate, quinine dihydrochloride and a host of other combinations of drugs.

Injection therapy fell into disrepute several centuries ago due to the untoward painful inflammatory processes set up by the extremely powerful sclerosing agents used. True, results were attained, but at too great a price. Consequently, the pendulum swung to surgical correction of the deformity to the exclusion of all other forms of therapy.

Within the past decade, however, a more rational approach to the use of sclerosing agents for injection has been made. The accumulated data obtained from varicose vein therapy have been applied to hydrocele. This is a condition which claims 2 to 4 per cent of all urologic admissions to clinics and hospitals, according to Ewell, Marquardt and Sargent.[1]

The late Dr. Norman J. Kilbourne and Dr. C. J. Murray[2] expounded the following requirements for the ideal injection solution:

1. It must not cause immediate pain at the time of injection.
2. It must not cause pain several hours after injection.
3. It must not cause undue local inflammatory reaction disabling the patient. He must not be kept from his regular work or lose time.
4. It must be bactericidal. If it is strongly bactericidal, the possibility of infection is eliminated.
5. It must not be dangerously toxic.
6. It must be efficient in destroying even large hydroceles with thickened walls, and yet it must not be so caustic as to bring on hemorrhage into the sac. This was reported in the old days with strong phenol concentrations.

After experimenting with eucupin, a quinine derivative, and quinine hydrochloride with urethane, Kilbourne added diothane as an additional anesthetic agent. It is this solution of quinine hydrochloride, urethane and diothane which forms the basis for our work. It was successfully applied in twenty-eight consecutive cases in the hands of Kilbourne,[3] but further confirmation was desired. At the suggestion of, and in collaboration with Dr. Clarence G. Bandler, who was one of the first to show the merits of diothane as an effective mucosal surface anesthetic agent in urol-

* Read before the Section of Genito-Urinary Surgery, New York Academy of Medicine, April 20, 1938.

ogy,[4] this work was undertaken. The fundamental technique of Kilbourne was followed.

NATURE OF NEW PREPARATION*

Let us consider the constituents and rationale for the inclusion of the three drugs mentioned in the injection solution under consideration. Quinine hydrochloride is an irritant that has bactericidal as well as anesthetic properties. This gives it preference over sodium morrhuate which is said to be non-bactericidal and painful on injection. The objection to quinine on a basis of idiosyncrasy or toxicity is a valid one. Auditory and optic nerve impairment has been reported after as little as 15 gr. of the drug had been taken. Since the content of quinine hydrochloride in this solution is only 5.5 per cent, a maximum of 10 c.c. would contain only 0.5 Gm. or 7.5 gr. The average dose used for injection in our hands was 6 c.c., representing 5 gr. No evidence of quinine sensitivity has been reported by any of the observers using this preparation.

Urethane is anesthetic in itself and also enhances the action of quinine, promoting its solubility, facilitating its penetration into the endothelial lining of the hydrocele sac, and most important, preventing a strong local inflammatory reaction. According to experimental work of Kilbourne, this latter property is an apparent specific action of urethane with quinine.

Diothane, a phenyl urethane synthesized by Rider,[5] has a powerful prolonged local anesthetic action and further enhances the effect of the solution. A noteworthy finding has been the total absence of pain from the injection per se. Naturally, rough handling of the scrotum and testicle will produce pain and striking the testis or cord structures with the needle during aspiration or injection may give excruciating pain. We have noted no post-injection discomfort.

Rider, of Merrell and Company, has provided a simple stable solution of the above mentioned drugs in the following proportions:

	Per Cent
Diothane	0.75
Quinine HCl	5.5
Urethane	3.0

The research product has been issued in 10 c.c. ampules, represented as a clear, colorless solution. Any discoloration in the solution should be a contraindication to its use.

The complete eradication of pain represents an accomplishment which has long been sought. Even in recent contributions to the literature we note references to this lone disturbing factor. Mayers,[6] in reporting on twenty-three successes using quinine hydrochloride and urethane states "an almost constant reaction at the time of the first injection is rather sharp pain." Baretz[7] reported twenty-nine, or 70 per cent cures in forty-two cases, with several patients complaining of pain at the time of injection of the quinine and urethane solution he used. While this pain never incapacitated, discomfort was experienced in some cases for one to two days. Kilbourne used quinine, urethane and diothane in twenty-eight cases without any pain and we have used it in twenty-six instances without such sequelae.

TECHNICAL PROCEDURE

Several points in the technique of aspiration and injection of hydrocele or spermatocele are worthy of note. The patient is placed in a supine position, the scrotal area prepared and surface antiseptic applied. We favor shaving the patient, both to combat infection and to facilitate application and removal of an adhesive suspensory. A small wheal is raised at the site of injection, using 1 per cent procaine hydrochloride. Kilbourne has emphasized the necessity for using a 3 inch 17 gauge needle for aspiration.

Various observers have advised special trocars and taking particular care in reversing the direction of the needle as it is pushed through the scrotal wall, thus

* The drug was supplied by the William S. Merrell Company of Cincinnati.

preventing leakage after its withdrawal and also helping to prevent it from slipping out of the sac. We followed this latter step, as advised by Kilbourne, in order to duplicate his technique exactly so that results could be evaluated. In recent cases we have simply used a 2 inch 19 gauge needle, inserting it almost at a right angle to the skin surface and directing it toward the upper recess of the sac—away from the testis and epididymis and parallel to the cord. We have noted no extravasation or leakage as a result. Care must be taken to keep the needle in place and we insert it, where possible, to its hub. In one instance, about 1 c.c. of the sclerosing agent was placed in the scrotal wall when the needle did slip out, but there was very little tissue reaction and no pain whatsoever.

Of great importance is the thorough evacuation of the sac. Depending on the size of the sac, a 10, 20 or 50 c.c. syringe may be attached to the needle for aspiration. Another 10 c.c. syringe filled with the drug is held ready for attachment to the needle. It is not sufficient to consider the sac empty when no more fluid can be aspirated. Digital palpation, "cornering" the fluid in the upper recess where the needle point lies, and manual expression without the syringe attached to the needle, will often yield 10 to 20 c.c. more fluid. It is readily understandable how ineffectual this amount of hydrocele fluid, if left behind, would render the sclerosing material.

Assistance makes the entire procedure easier, but we have carried it out single-handed, in both the clinic and the office. Depending on the amount of fluid obtained, a proportionate volume of the drug is injected. Thus, 2 c.c. was injected into a sac from which 15 c.c. of fluid was obtained; 300 c.c. of fluid was replaced by 10 c.c. of the drug. The dose is arbitrary—the aim being to inject enough to reach the entire endothelial lining of the sac. This brings up another important step in technique—that of "kneading" the collapsed sac following injection, to facilitate uniform distribution of the drug.

Finally, we favor a measure which has not been employed or emphasized by others. We apply a snug, adhesive suspensory immediately after the injection. This is kept on for three to five days without being disturbed. Following its removal a bag suspensory is worn until cure is effected. We believe the immediate compression of the collapsed sac enhances the action of the drug on the endothelial surface. Further, after the first week, nightly hot sitz-baths are advised to promote speedier resolution of the fluid accumulation.

ANALYSIS OF CASES

Our particular study embodied a total of nineteen hydroceles and seven spermatoceles in twenty-three patients, whose ages ranged from 4 to 75 years. Thirteen, or 60 per cent, were over 50 years of age. Fifteen patients, representing thirteen hydroceles and four spermatoceles, have been followed through to apparent cure and have been observed for follow-up periods varying from one to fourteen months. It is this group which forms the basis for our observations and conclusions.

Some of our cases represented clinic material, and because of the migratory nature of the adult male patient, we have seven incomplete records. We wish to express our indebtedness to Dr. J. A. Hyams for making such material available in the Urologic Out-Patient Department of the Gouverneur Hospital, and to Doctors D. A. Anderman and S. Klein for their clinical assistance. Private cases are also included in the study.

A brief analysis of the seventeen completely followed apparent cures in fifteen patients yields some interesting data. In all except one case, definite etiology could not be elicited and they were therefore classified as idiopathic. One followed partial phlebectomy for varicocele. The size of the hydrocele or spermatocele as determined by fluid content varied from 4 to 300 c.c. and the amount of quinine hydrochloride, urethane and diothane injected ranged

from ¾ c.c. to 10 c.c. The cases were consecutive and unselected.

Injections were made from one to four weeks apart. The number of injections necessary for final eradication of the sac was: in nine cases, one injection; in five instances, two; and three, four, and five injections, respectively, were necessary in three remaining cases. In the early cases we were too anxious to effect a rapid cure. We have since learned to wait at least three weeks between injections, for little is gained by repeated rapid injection therapy. We feel certain that the next group of cases in this series will show far fewer injections per case.

The aspirated fluid is routinely examined microscopically. The hydroceles yielded clear yellow fluid on initial aspiration and subsequent post-injection specimens were either clear, hazy or deep orange in color. Spermatoceles yielded pathognomonic milky fluid. Following injection and with diminution in the fluid content of the sac, a characteristic crepitant sensation is elicited, not unlike a pleural friction rub. This finding is evidence of effectivity of the drug and bodes well for ultimate cure. No infection, local or systemic reaction was noted in any of the cases following injection therapy. Several workers have cited this point as a contrast to operative figures published by Campbell[8] in reviewing 502 open operations for hydrocele. He noted a 15 per cent incidence of superficial or deep infections. Six patients developed uninfected and twelve infected hematoceles, eight of the latter resulting in orchidectomy. Despite a total of thirty-one injections, we did not have a single case of hematocele.

Of the seven cases listed as incomplete, three did not return to the clinic and investigators could not locate them. Two were more recently injected. Two represented communicating hydroceles, ordinarily a contraindication to injection therapy. Care was taken, by using pad compression over the inguinal canal to prevent entry of the solution into the peritoneal cavity.

Injection in these cases was carried on from a purely academic standpoint, since operation was indicated and we were seeking histologic studies of the effect of the sclerosing drug on the endothelial lining of the tunica. Cures were not anticipated nor effected, but the patients have put off surgery—unfortunately. We have not classified these cases as true failures since we could not consider them suitable ones for injection therapy and would not have resorted to this course except with the hope of obtaining tissue studies.

SPERMATOCELE THERAPY

Of four spermatoceles, three were cured by one injection. A characteristic induration remains, varying from pea to olive-size. It apparently represents a contracted sac, for attempts at aspiration of possible fluid content have been unsuccessful. In two instances, the sacs were loculated and as many as three individual cavities on one side have been aspirated and injected at one sitting. Others have reported indifferent results in injection therapy of spermatocele. Our own few results are gratifying since the tendency of the lesion to recur even after surgical excision is well known unless the epididymis and rete testis are sacrificed.

An interesting observation was made in studying the fluid content of the spermatocele microscopically, before and after injection. Prior to treatment, the fluid contains swarms of actively motile spermatozoa. Subsequent examinations after injection show either very sluggish motility or complete cessation of activity and a diminution in numbers. The spermicidal action of the drug may explain this efficacy.

A possible explanation of the mechanism by which sclerosing solutions act has been advanced by Ewell, Marquardt and Sargent.[1] A patient who had been injected three times with quinine hydrochloride and urethane and cured, was prevailed upon to submit to operation one month later. Grossly, a normal testis and epididymis were found, together with a small amount

of dark amber fluid and several long loose strands of attached and organized fibrin. Microscopically, the endothelial layer was intact, the blood vessels appeared normal, but the subserous tissue of the tunica was distinctly thickened and infiltrated with organizing fibrous tissue. These workers advanced the hypothesis that fibrosis of the tunica wall after injection so interferes with the blood and lymph supply of the endothelium that it alters the process of fluid formation or absorption.

Others feel that obliteration of the sac is accomplished by adherence of apposing walls following a sclerosing inflammatory reaction. While this is apparently a logical theory, the sparse work on the subject does not bear it out. Further study along such lines is essential.

Without question, injection therapy is an ideal procedure for eradication of chronic hydrocele or spermatocele in the aged, the poor operative risk, in recurrent postoperative hydrocele or spermatocele, in hydrocele following partial phlebectomy for varicocele, in those who refuse surgery but will submit to office injections, and for that large group which, for economic reasons, cannot be hospitalized.

CONTRAINDICATIONS

Contraindications to injection therapy apply to the acute hydrocele which will usually subside with rest and immobilization, and to communicating hydroceles with or without hernia. It has been stated that without open operation, neoplasm or tuberculosis may be overlooked. If careful palpation is made of the scrotal contents when the sac is first emptied, such errors in diagnosis would not occur. Needless to say, all cases suspected of possible epididymal or testicular lesions should be explored.

With the advent of a nontoxic, nonirritating, effective sclerosing agent, such as quinine hydrochloride, urethane and diothane whose injection is free of pain, many of the previously reported complications of injection therapy of hydroceles may be obviated. Local and systemic reactions, slough, infections, injury of testicle, epididymis or cord structures, and hematocele must now be attributed to faulty technique or to the drug. To avoid injury to a large scrotal vessel at the time of injection, we suggest the added precaution and refinement of placing a lighted electric light under the sterile draping at the time of injection to transilluminate the mass. We have used a Cameron light which can be sterilized and brought in direct contact with the scrotum.

The relative merits of aspiration and injection as opposed to open operation can only be determined by careful statistical comparison. Sanford[9] noted thirty, or 2.4 per cent relapses, in 1,216 cases of open surgery for hydrocele. Of these, there had been late investigation of 412 cases with twenty-two relapses, or 5.33 per cent. The same author revealed that 6 to 14 per cent failures had been reported following injection therapy, although conceding that with newer methods and drugs the percentage of failures is lower.

In evaluating recurrences after injection therapy, one must distinguish between fluid re-accumulation which may require one or more injections to eradicate, and true return of fluid after a period of apparent obliteration of the sac. Nor should one call a case a failure which does not respond to a single injection of the sclerosing solution.

CONCLUSIONS

We are presenting our data as a preliminary report, with final evaluation deferred, pending wider application of the method in our own hands and those of others. No complications, disability, or recurrence to date has occurred—admittedly a promising beginning.

The economic status of the patient is an important factor in the choice of therapy. Inability to face the expense and time loss involved in surgical treatment of hydrocele may counterbalance the inconvenience of a series of injection treatments over a period of time. In charity hospitals, where beds

are at a premium, injection therapy is an invaluable therapeutic aid in the outpatient department.

The simplicity of the method may prove its worst enemy, for its indiscriminate use by those inexperienced in its technique or unable to recognize scrotal pathology will only bring discredit to a procedure which time and experience may prove to be a method of choice. The best test of any procedure lies in whether one would subject himself to the same method if he had such a condition. For myself, I would certainly have a hydrocele injected before choosing open surgery.

Sincere thanks are extended to Dr. Clarence G. Bandler for his sponsorship of this work and and the privilege of making this report.

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DIRECT AND INDIRECT HERNIA IN THE LIGHT OF THE NEWER INTERPRETATION OF THE ANATOMY OF THE ANTERIOR ABDOMINAL WALL*

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IN a former contribution, the following concepts were advanced in reference to the inguinal region:

1. The primary cause of hernia is the so-called opening through which the viscus appears, and not the viscus per se. The opening should not be considered as a defect nor fault in the body wall, but rather as a definite well-planned exit for structures, which through evolutionary changes have been found to be most advantageously maintained outside the abdominal cavity.

2. In a comparison of the inguinal canal with the many diverse valvular constructions found elsewhere in the body, one is impressed with the singleness of pattern or uniform mechanism employed, a fact which heretofore has not been sufficiently stressed in the literature. The mechanism is the oblique projection of the structure to be constricted through two or more layers of muscle, the valve effect or constriction being obtained from the muscle tone or by muscle contraction. This may be considered as a universal scheme of valve construction.

3. The average pattern of the inguinal region, or the lateral division of the abdominal wall, is directly dependent upon the development, which may be considered as having two factors: (a) the development of the abdominal plate; and (b) the development of the funiculus. The insertion of the gubernaculum cord, i.e., the funiculus non-growth factor, in the undifferentiated abdominal wall and the subsequent development about it of three parallel muscles, two active and one passive, give evidence of such a valvular construction. These three muscles by their fan-like insertion, slide action and contraction, normally close the triangular space bounded by the ilium, pubis and the Poupart line. Little importance is placed in the pressure resisting action of the external oblique muscle, which because of its fixation and early canalization plays only a passive rôle in the resistance to intra-abdominal pressure.

4. Conceiving nature as continually experimenting (or as some call the process, evoluting), one naturally comes to the thought that this evolution process causes changes, i.e., variations in the primary pattern of valve construction. These changes of variation, however slight they may be, will influence (a) the definite pattern, (b) the size, shape, and tension of exits, and (c) will produce widened, narrowed, and attenuated conditions of the transversalis and internal oblique muscles, the structures producing the exits. The depicted variations are due to excessive pull or recession of these muscles from the Poupart line. Such modifications result in a greater than ordinary triangular space between the ilium, pubis, and Poupart line, and leave the space unprotected to a greater or lesser degree from the intra-abdominal pressure, thus enhancing the potentiality for hernia, i.e., the protrusion of a viscus.

In my earlier paper I stated that the important problem is to determine whether inguinal hernia, direct and indirect, occurs more frequently in the cases in which the triangular space bounded by the ilium, pubis and the Poupart line is largely unprotected. At the outset of the present

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investigation it was realized that this problem and the concepts of my former contribution are but a division of a greater problem—the consideration of the entire anterior abdominal wall, especially that part which is infra-umbilical. This has to do with the divisions of the abdominal wall; the average pattern; the variations from the average pattern, and the effect of the variations on the potentiality for hernia. Of further importance is the question, what rôle, if any, does the reciprocal compensatory interplay of the divisions of the anterior abdominal musculature play in the prevention of hernia?

For purpose of analysis the anterior abdominal wall may be divided into two parts: a *medial,* bounded by the linea semilunaris and ventral midline, the lower rib margin, and the pubic bone; and a *lateral,* bounded by the linea semilunaris and the dorsal flank, the lower rib margin, and the ilium and pubis. For the purpose of description the anterior abdominal wall may be considered under three heads: (a) the development of the mesenchymal, blastemal or myotomal plate; (b) the development of the gubernaculum cord and the spermatic funiculus; and (c) the development of the rectus abdominis muscle cylinder.

The anterior abdominal wall begins as a ventral extension of the thoracic myotomes that fuse into an undifferentiated mesenchymal or blastemal plate, in which are developed the ribs (seen in embryos of 5 to 7 mm.). The rib rudiments seemingly divide the plate (which is lined within by undifferentiated endoabdominal fascia and peritoneum, and without by undifferentiated external body fascia) and skin into two tangentially splitting sheets—a *lateral* (which differentiates into the posterior serrati muscles and external oblique muscle), and a *medial,* which extends between the ribs and medial to them, forming the intercostal muscles, the internal oblique and transversalis muscles (seen in embryos of 14 mm., 7 to 8 weeks). The external oblique, internal oblique and transversus muscles become parallel and superimposed and extend ventrad to the midline, caudad to the upward progressing coxal blastema and the blastemal inguinal ligament, being guided to contact by the ventral curving of the ribs and the formation of the sternum. Toward the eighth or ninth week (18.5 to 20 mm. embryo), the symphysis pubis is formed (at first composed of blastemal tissue, in which tissue, first hyaline, then fibrocartilage, becomes differentiated).

After completion of contact by the downgrowing abdominal plate with the coxal bone and the inguinal ligament, the true pelvis is brought into the upright position from the horizontal forward position, by the enlargement of its sagittal diameter, causing the frontal-facing pubic crest to rotate to its definite position, which is superior and posterior. The dimensions of the differentiating mesenchymal abdominal plate then are from the flank to the vental midline, and from the lower costal rim above to the ilium-pubis and the symphysis below. An intensification of growth occurs at this time (8 to 9 week, 20 mm. embryo). This growth episode in the musculature of the abdominal plate causes a pull or stress in three directions, sagittal, longitudinal and coronal. As a result of this tridirectional growth and stress, the abdominal muscle plate, which is being tangentially split into three superimposed muscles, undergoes transformation or degeneration into fasciae and aponeuroses, at areas of maximum tension, causing a recession of the muscle fibers along the inguinal ligament, pubic bone, and midline, which are the areas of maximum tension. Because of this transformation or degeneration, only the fascial coverings of the muscles remain in these regions and there are formed areas of muscular deficiency, the size of which depends upon the intensity of the pull or stress and the amount of degeneration. These areas have heretofore been considered defects in the abdominal wall. Since the line of muscle tension is 90 degrees it will cause the areas of muscular deficiency to be semicircular in shape, and cause the forma-

tion of arch pillars or points of retention. The arch spans are (1) ventromedial arch, linea semilunaris (condensation of the external and internal oblique and transversalis muscles), from the costal cage to the pubic bone as the lateral margin of the sheath of the diameter increasing rectus abdominis cylinder; (2) inguinal arch from the pubic bone to the inguinal ligament, as the fibers of the transversalis and internal oblique muscles; (3) arch of Douglas, from the pubic bone to the symphysis, as the arching fibers of the transversalis muscle.

There is formed by the union of a pillar from each of the three arches of this multiple arch formation a common pillar of retention or insertion into the pubic bone, the conjoined tendon. The anterior abdominal wall acts as a contraction mechanism, about the inserted structures, i.e., rectus abdominal muscle cylinder and gubernaculum cord found in the above described areas of muscular deficiency, and effectually closes the areas, provided that all essential structures of the abdominal wall adhere to the primary pattern and no variants develop. The inserted structures aid in the closure by their own contraction and their increase in diameter.

In this seemingly solid tangentially laminated plate of fused myotomal tissue, extending from the dorsal thorax to the ventral midline and from the lower rib margin to the ilio-inguinal ligament and the pubic bone, two special structures appear, even before differentiation of the myotomal mass. *First*, the gubernaculum, lateral in position and at right angles to the inguinal ligament and the muscle plate or sheets, and *second* the rectus abdominis cylinder. The gubernaculum cord, consisting of a group of special muscle cells arranged into a cylindric mass, joins the inguinal crest and the testicular ligament on the inside and the scrotal ligament and skin on the outside to become a completed structure, the gubernaculum testis. In the differentiation of the abdominal plate, the two inner muscles, internal oblique and transversalis seem to enfold the gubernaculum cord, while the third or external oblique becomes evaginated for its exit.

Fig. 1. Transversus abdominis muscle is fascial. Internal oblique, while high arching, is fleshy. Conjoined tendon is long but of good quality. No hernia present, but a potential type.

Fig. 2. Transversus abdominis muscle is fleshy with cephalic recession above the anterior superior spine. Internal oblique is of average pattern and fascial. Conjoined tendon consists mainly of transversalis component. A potential hernial type.

The longitudinal growth of the body causes the muscle fibers of the internal oblique and transversalis to be seemingly pulled from the Poupart ligament into a fan-like movement, resulting in the attenuated fascias of the internal oblique and transversalis muscles. This process is accomplished by muscle growth and the growth of the ilium; as a result, therefore, of the cephalic movement only the fascial covering of the internal oblique and transversalis muscles remains. This results in an area of muscle deficiency bounded by the ilium-pubis and Poupart line, the size of which depends upon the amount of recession of the muscle fibers. Thus is formed what is considered by some a defect but in reality is a point of exit for the testicle. Because the gubernaculum cord has no cephalic growth, the testicle is apparently pulled down, but in reality it is guided into the evaginating attenuated fibers and fascias of the internal oblique and transversalis muscles, retaining them as coverings. These coverings are collectively called the cremaster body. When development is complete, the funiculus acts as a substitute for the pressure-resisting muscles, i.e., the internal oblique and transversalis in the triangular arch space bounded by the ilium-pubis and Poupart line.

Fig. 3. Variation of the transversus muscle with poor compensation of the internal oblique. Result—a direct hernia.

Fig. 4. An indirect hernia due to fascial degeneration of transversus and internal oblique muscles. Conjoined tendon components are completely fascial.

At about the sixth week (7 to 9 mm. embryo) while the abdominal plate or sheet is still indefinitely laminated into three superimposed muscles, there differentiates in the advancing edge of the myotomal plate (the myotomal lateroventral downgrowth) a group of special muscle cells that are arranged at right angles to the axis of the growth, and form a cylindric mass, the rectus abdominis rudiment. The above cylindric mass of tissue is formed from the components of both the *medial* sheet (that differentiates into the intercostals, internal oblique and transversalis muscles), and the *lateral* sheet (that differentiates into the external oblique and the posterior serrati muscles), but mainly from the medial sheet. It causes to be formed about it an oval cylindric tube or condensation of tissue or sheath which is derived from both sheets of the myotomal plate. The lateral line of demarcation of the sheath is called the linea semilunaris. The rectus rudiment, there-

fore, extends from the outer fascial limitation of the mesenchymal plate (the ecto- or external abdominal fascia) above, to the inner fascial limitation (the endo-abdominal fascia), below.

Fig. 5. Transversus and internal oblique muscles apparently show an average pattern, but are really variations of marked degree in that their fibers split, causing condition of marked recession and hernia potentiality and formation. The conjoined tendon is fascial, showing degeneration of the medial aspect of the muscles with poor resistance to intra-abdominal pressure. Result—hernia.

Fig. 6. A right undescended testicle. Obstruction caused by no arch formation of the lateral component.

As and when (seen in a 14 mm. 7 or 8 week embryo) the curving lateroventral downgrowth makes contact with the coxal bone, the ventromedial part containing the rectus abdominis rudiment, makes contact with the boundary of the true pelvis at the pubic rudiment—the pubic rudiment having been brought into the upright position from the horizontal forward position by the enlargement of the sagittal diameter of the true pelvis. After the rectus abdominis rudiment makes contact with the pubis, it extends from the anterior rib aspect, beneath the anterior limitation of the lateral sheet, the ectofascia, to the pubic crest insertion and posterior aspect or limitation of the medial sheet, the endofascia, and is enveloped by the internal oblique and transversalis muscles, as is the gubernaculum cord and its definitive funiculus spermaticus.

We may now visualize an average pattern of the anterior abdominal wall. It is a flat, musculofascial structure consisting of two major divisions, a medial division and a lateral division united by a curved fascial condensation or reënforcement, the linea semilunaris or ventral arch, which is the lateral demarcation of the rectus abdominis sheath, and extends from the costal margin to the lateral aspect of the pubic bone.

Each of the divisions is partly muscular and partly fascial or aponeurotic. In the caudal part of each is found an arch of muscle or aponeurotic fibers, the inguinal arch (internal oblique and transversalis muscles), and the semilunar arch of Douglas (transversalis muscle), respectively, from the concave edge of which there is prolonged to the attachment on the coxal bone attenuated fasciae, the result of transformation or degeneration of the laminated superimposed muscles. The medial pillars of these two arches fuse with the caudal extremity of the fascial reenforcement, the linea semilunaris, to form

a structure of insertion into the lateral part of the pubic bone, the conjoined tendon. The lateral pillars of the arches are inserted, respectively, into the ilium pubic line (inguinal ligament) and the symphysis pubis. A bridge of muscle and fascia extending from the ilium (anterior superior spine) to the midline (symphysis pubis) and buttressed by a medial pier of support, the conjoined tendon, is the result; and as a bridge construction reacts to stress, so, too, the entire abdominal wall and not only any one part or division of it will react to intra-abdominal pressure.

The mechanics of bridge construction make evident the compensatory action of parts of the abdominal wall under pressure, and as a corollary there follows this fact, that the shorter the distance from pillar to pillar of an arch or series of arches, the greater the strength found in the bridge construction and the more resistance it has to strain. When the arches and the areas of muscular deficiency are of average pattern and no variants develop in the wall, the insertions of the gubernaculum cord or the definitive funiculus spermaticus and the rectus abdominis muscle, about which the arch formations contract, are accessory factors and are of but secondary importance in the resistance to intra-abdominal pressure. We are also brought to the thought, that as a bridge consists of many parts or members, not all of which are of equal tensile strength, the ability to stand strain, even excessive strain, without failure is due largely to the compensatory shift or stretching that occurs under stress; so too, in the mechanics of the abdominal wall there is a compensatory action of parts of the abdominal *wall*, and even when there is excessive stress upon a greatly strained area, complete failure of resistance to the intra-abdominal pressure or hernia, i.e., viscus protrusion, is prevented.

In the former study the transversalis muscle was considered and variations depicted. In this study, comparative in nature, it was found that while the abdominal wall acts as a whole in the average pattern, variations are found in each component of the abdominal wall. These variations influence the coördination of its component parts and its potentiality for hernia. The reasons for the small number of hernias generally found where the areas of muscular deficiency of the abdominal wall are greater than average, is the ability of one part to compensate for another.

In a detailed study of variations of the abdominal wall, it becomes necessary to consider each individual component, and to analyze its effect in minimizing or aggravating the potentiality for inguinal hernia, direct and indirect. Accordingly, I shall describe the average pattern and thereafter the variational types.

The fibers of the transversus abdominis and internal oblique muscles that hug the cord snugly are arched from the medial one-third to one-half of Poupart's ligament to the lateral aspect of the pubic bone. The muscle fibers having degenerated or transformed or receded synchronously and equally, the tilting of the pelvis causes a seeming difference in elevation. In the variations may be found recessions of all degrees, from absence of recession (where due to defective tension lines the muscle fibers fail to recede from Poupart's ligament or to form an arch), to recessions measuring 1½ inches above the anterior superior spine. Such variations of the arching fibers are found in both muscles, the internal oblique and the transversus, being frequently present as a variant in one, with an average pattern in the other.

While excessive recession of the muscle fibers and the production of areas of attenuated fasciae of greater than average size, at the sites of maximal tension, is frequent, the opposite condition, i.e., absence of recession, is not infrequent; the latter being due either to faulty angles of tension or to total absence of the amount of pull or stress which is required to produce an average pattern of the anterior abdominal wall. As a result the muscle fibers do not recede from the coxal bone; form no arches or bridge of muscle fibers

and no areas of attenuated fasciae. Absence of recession or arch formation indicates an arrest of development at the seventh or eighth week (the 14 mm. stage), and since it is at this time that the change in position of the testicle known as the descent of the testis begins, any arrest in the development of the abdominal muscle plate will influence this mechanism. Due to the arrest in development of the muscle plate the gubernaculum cord, while present, is prevented from functioning, and the larger testicle meets resistance at the inner aspect of the abdominal wall and finds its exit through the wall and into the scrotum blocked. The cases of delayed descent may be explained by a late or tardy recession of the fibers from the coxal bone at the time of puberty, at which time there occurs a marked longitudinal growth episode.

The conjoined tendon is usually described as those parts of the internal oblique and transversalis muscles which unite to insert simultaneously into the pubic bone (crest and tubercle) in front of the rectus abdominis muscle. There is no fault to be found with the first part of the above description, but we cannot, in view of the foregoing, consider the conjoined tendon as a simple simultaneous union for insertion. It is rather the middle pier of the anterior abdominal wall with a definite construction the purpose of which is resistance to intra-abdominal pressure. Variations indicate that its types are long, short, widened and attenuated. These types may also be considered as indices to the variations of the laminated muscles with which they are continuous. Therefore, the conjoined tendon is found to be completely muscular or completely fascio-aponeurotic, depending upon whether the muscles adhere to the average pattern or are variants.

The rectus abdominis muscle being a simultaneously developed structure will be affected by the same growth mechanism as is the abdominal wall and will, therefore, show the same types of variation. The lower part due to strain will be muscular or fascial to a greater or lesser degree depending upon the stress put upon its musculature after completion of contact with the pubic bone. Thus we find short, bulky, muscular and long, attenuated, tendinous insertions of the rectus abdominis muscle. These insertions, located in areas of attenuation of the anterior abdominal wall that are greater than average, will influence the potentiality for hernia in direct proportion to the amount of muscle, or fascial content found in the insertions. The more muscle, the greater the ability to contract and increase the diameter of the rectus, the greater the ability to aid in the protective mechanism required to resist intra-abdominal pressure.

SUMMARY

1. A detailed study and a completed picture is drawn from the available embryologic data describing the anterior abdominal wall.

2. The average and variational types of the component parts of the abdominal wall are detailed and depicted, and their effect upon potentiality for hernia analyzed.

3. There are advanced:

(*a*) An explanation of the mechanism of formation of the linea semilunaris.

(*b*) A reason for the belief that one of the causes of non-descent of the testicle is mechanical obstruction.

(*c*) A concept of the anterior abdominal wall as a bridge construction of multiple arches, the mechanism and function considered as potential factors in the causation of hernia.

(*d*) A concept of the conjoined tendon as the middle pier of support of the anterior abdominal wall.

(*e*) A reason why more hernias do not occur when the area of muscle deficiency is largely increased from the average.

(*f*) An explanation of reciprocal compensatory action of the entire abdominal wall as a preventive of hernia.

CONCLUSIONS

1. Complete failure of the abdominal wall in its resistance to intraabdominal pressure and hernia formation is improbable, if not impossible, when an average pattern is present.

2. Hernia formation may occur as a structural and functional failure of the entire abdominal wall or of any part or division due to:

(*a*) Deficiency in the musculature of the component parts of the abdominal wall, the result of extreme degeneration or transformation into fasciae or aponeuroses, which have little ability to resist pressure.

(*b*) Variations in the component parts of the abdominal wall of such degree that complete valvular closure even with maximum contraction is impossible.

(*c*) The occurrence of simultaneous variations in two or more components of the wall.

(*d*) Delayed or non-coordinated action of the components of the abdominal wall, resulting in a failure of compensation and a deficient valvular mechanism.

This paper has as its purpose an attempt to clarify some of the basic factors that underlie the production of hernias, direct and indirect, and the causes of postoperative recurrences of direct and indirect hernias. The phases studied and detailed have been those of the variations from the average pattern.

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TEN YEARS' EXPERIENCE WITH LEG ULCERS IN THE OLD AGE GROUP*

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WE present a statistical review of leg ulcers based upon the material of the last ten years at the Neurological Hospital. The number of these cases was so great that this division of the surgical wards could be distinguished with ease by the olfactory sense. As a result, these cases were in favor with neither the physicians nor the nurses. Each patient was furnished with a pail containing hot water and soap, some ointment and bandages—he treated himself! Only in the Home† were the patients "doctored." An inmate and former sailor had opened his "office" behind the firehouse in a shack where he conducted a well frequented "clinic."

The surgical department started its work by eliminating this eyesore. An ulcer program was organized on a scientific basis. Consequently, the once familiar scene of the aforementioned privileged patient holding his "clinic" in full "septic" sway, was no more. We organized an ulcer service from the scrap, assigning the member of the surgical staff most experienced along these lines to the care of the cases. This practice has been continued up to the present time.

By frequent consultations and reëxaminations, interest in these cases has been kept awake and abreast of modern scientific developments. To the old and approved method of compression bandages which we have used routinely, new therapeutic measures are continually being added to our armamentarium. This selection is being made according to sound surgical principles out of an abundance of methods which in themselves sometimes pay no attention to surgical principles and herald methods as new, which in reality were generally adopted for more than half a century. For this reason, we consider it appropriate not to restrict ourselves to mere figures and statistics, but also to present a brief clinical picture of our cases including treatment and diagnosis in general.

Our statistics cover the period from January, 1927 to December, 1937. During this decade, we discharged from the hospital 134 cases of leg ulcers. The records of all these cases were carefully studied and included the nurses' notes for completeness.

Classification of the cases according to treatment proved impossible. Whereas ace and elastoplast bandages or Unna boots were used routinely in all cases, the additional auxilliary treatment was selected not only for each case individually, but also modified and adapted in each case to the changing condition of the ulcers as well as to new drugs and the new means that scientific research was offering at the time. Therefore, in addition to the pressure

† The Home is the city almshouse of approximately 1,700 inmates, who when medical care or hospitalization is required are transferred to the adjoining Neurological Hospital. These institutions are located on Welfare Island, New York City.

* The material in this article was submitted as part of a symposium on leg ulcers presented at the Neurological Hospital January 6, 1938. Included discussions were Anatomy, Physiology and Etiology by Dr. B. A. Goodman and Special Technique by Dr. H. J. Philipp.

bandage, a variety of treatments has been used. This point is illustrated in the following cases:

Mr. J. K., 80 years old when discharged, had been treated in the hospital for five years for an extensive ulcer with ace bandages, Unna boots, numerous ointments, insulin dressings, diathermy, histamine injections to improve arterial circulation, *x*-ray, and curetting of the ulcer.

Another patient with a large ulcer was turned over to the physiotherapy department for histamine iontophoresis. Later he was found to have sarcomatous degeneration of the ulcer which necessitated amputation.

C. G., 63 years old, had an extensive ulcer which required skin grafting as an auxilliary means after the development of sufficient granulations.

The cases cited demonstrate the necessity for surgical supervision and follow-up as well as the impossibility of classifying our material according to treatment.

Pathologic classification is more feasible with our material and will be outlined subsequently. Before discussing the statistics we present a general view of our material.

There were 134 cases treated in all over the full period from 1929 through 1936. Of these only nine were below the age of 50, while the vast majority were over 60 years of age. The age distribution is shown in the following table:

	Number	Per Cent
Below 50	9	7
50–60 years	32	24.8
60–70 years	47	36.5
70–80 years	35	27.1
Over 80	6	4.7
Unknown	5	
Total	134	100.0

The largest group of patients is that between 60 and 70 years of age, the smallest the group over 80.

In patients of this age group it is to be expected that concomitant diseases would be of frequent incidence. Syphilis, heart disease and arteriosclerosis were the most frequent contributory disease factors. Cardiac conditions, particularly chronic myocarditis and valvular disease, were diagnosed in not less than seventy-four cases (55.2 per cent), and general arteriosclerosis in almost as many. It is our opinion that the figure for arteriosclerosis might well be corrected, since probably 65 per cent of the patients suffered with it. Among these arteriosclerotics, every tenth patient developed gangrene of the affected leg and every twelfth patient a cerebral hemorrhage. In thirty-five cases arteriosclerosis and heart disease were associated, and this group showed the highest mortality.

Table I
CONCOMITANT AND CONTRIBUTORY DISEASES

	Number	Per Cent	Per Cent Mortality
Syphilis	11	8.2	
Cardiac	74	55.2	43.2
Arteriosclerosis	60	44.8	33.3
Cardiac plus arteriosclerosis	35	26.1	48.5

The mortality rate in general proved rather appalling: fourty-four of the total number—every third patient—figured in the mortality statistics. However, it must be emphasized that the age group was very high and that in no case had the cause of death any connection with the leg ulcer.

Table II
RESULTS OF TREATMENT

	Total Number of Cases (134)		Completely Treated Cases (93)	
	Number	Per Cent	Number	Per Cent
Completely healed	38	28.0	38	40.0
Almost healed	29	21.6	23	24.7
Greatly improved	35	26.1	23	24.7
Improved	12	9.7	6	6.4
Not improved	20	15.0	3	3.2

The death rate of the cardiac-arteriosclerotic group was higher than the general average.

For the entire group of 134 cases, the results of treatment are given in Table II. They show that good results were obtained in almost half the cases, and some improvement in 85 per cent. In the ninety-three cases in which treatment could be carried through to its conclusion, the results were much better: good results in 64.7 per cent and improvement in 96.8 per cent.

The effects of treatment as related to concomitant diseases is shown in Table III, both for the entire group and for those cases in which treatment was completed.

Table III
Results of Treatment

	Cardiac (74)	Completely Treated (45)	Arteriosclerosis (60)	Completely Treated (41)	Syphilis (11)
Completely healed	24.3*	40.0	28.4	41.5	
Almost healed	20.2	24.4	23.3	22.0	18.1
Greatly improved	25.6	26.6	21.6	24.3	45.5
Improved	14.8	6.6	11.6	7.3	27.2
Not improved	14.8	2.2	15.0	5.0	9.0
Died	43.2	....	33.3	....	27.2

* All figures on percentages.

If the facts presented regarding the age, contributory diseases and death rate of our series are pictured collectively, we have, to the best of our knowledge, an unparalled group of cases yet to be published. A pessimistic attitude regarding the curability of any one case is readily understandable.

In the cases complicated by syphilis, eleven in all, none was completely healed; 18.1 per cent were "almost healed" on discharge; 45.5 per cent were greatly improved; 27.2 per cent were somewhat improved, and 9 per cent were not improved. The cardiac group was the largest, seventy-four cases. Of these, 24.3 per cent were discharged "completely healed," and 20.2 per cent "almost healed." "Good results" represent 45.5 per cent of the cardiac cases. The arteriosclerotic group, slightly smaller than the cardiac, with sixty cases, showed 28.4 per cent "completely cured"; 23.3 per cent "almost healed," that is, "good results" in 51.6 per cent of the cases. The failures are almost identical with the fatalities. The death rate among the arteriosclerotic failures was 80 per cent; among the respective cardiac group 90 per cent.

In no case in the whole series had death been caused by the ulcer. In fact, the ulcer in a great number of the patients was only an accidental finding. We therefore feel that our results are distorted, and that it would be fair to include in our statistics an evaluation of the cases in which we had the opportunity to carry out the treatment for a reasonable length of time. Among forty-five cardiac cases in which treatment was carried through, 40 per cent were discharged "completely healed"; 24.4 per cent "almost healed" or 64.4 *per cent with "good results."* Among the forty-one arteriosclerotic cases, 41 per cent were discharged "completely healed," 22 per cent "almost healed"—i.e., *"good results" in 63.5 per cent of the cases.*

The excellent results obtained have considerably altered the aspect of our wards. Whereas formerly leg ulcers belonged to the category of common diseases, there are no more than six cases in the hospital at present and about thirty cases in the Home (all of them ambulatory).

The varicose type of ulcer has predominated, although the arteriosclerotic type is also very common. Several syphilitic and occasional neoplastic ulcers have been seen. Other types include the trophic, infectious, tuberculous, traumatic, epidermophytotic, actinomycotic, and ulcers due to other granulomata.

Etiologically, the one outstanding factor is that the great majority of leg ulcers (93 per cent) occur after the age of 50. Pathologically it is evident that they are frequently associated with arteriosclerosis of the vessels of the legs. (Fig. 1.) This latter relationship is of considerable importance, since we have been impressed with the frequent finding of arteriosclerotic changes in the vessels of the lower extremities in autopsies of patients who had

chronic leg ulcers. Occasionally the arterial changes are found to be much further advanced than the changes in the veins.

Fig. 1. Small artery at the periphery of an ulcer with marked arteriosclerotic changes.

The varicose ulcer is essentially a chronic ulcer. Its chronicity may be maintained by several factors: (1) chronic irritation; (2) infection; (3) poor nutrition, including trophic disturbances; and (4) circulatory insufficiency. All these factors may, and frequently do, occur simultaneously. But the determining cause in most cases is eventually a circulatory one, for in the presence of an ample circulation the other causes may be obviated.

Due to pressure from above, or from the force of gravity exerted after prolonged standing, the deep veins lose their competency. As a result, the blood, instead of flowing from the superficial veins into the deep ones, at times flows in the reverse direction. Stagnation of the blood in the superficial veins occurs, and is followed by a general retardation of the blood stream, so that finally the walls of the veins themselves receive insufficient circulation for their needs. These walls weaken, and fluid begins to exude from them into the neighboring tissues. Because of the intense congestion, some of the small veins rupture, discharging their contents into the tissue spaces. Some of this blood is absorbed, but part of it remains as blood pigment. (Fig. 2.) Due to the presence of the latter as well as to the occurrence of occasional trauma and infection, an inflammatory process is set up. Healing takes place slowly with resultant connective tissue deposition which in contraction causes obstruction of the small blood vessels and lymphatics. Obstruction in the latter causes further edema in the tissues, while obstruction of the former results in poor nutrition and further tendency to ulceration.

Thus a vicious cycle is established. The small arterioles are continually disturbed due to accumulation of cells in the inflammatory process and become sclerosed or thrombosed. The venules remain dilated. The surrounding tissue is edematous. Ulcers come and go. At first the epithelium is able to regenerate and cover the healing ulcer, but as the process continues and the blood supply of the arterioles is diminished, regeneration becomes impossible. If now the larger arterial trunks have become arteriosclerotic, the chronicity of the ulcer is established. Whatever healing does take place, occurs by fibrosis after the superficial tissue has been sloughed.

Particular emphasis must be laid on the scarring process which may be extensive enough to involve all the tissues in the vicinity of the ulcer, extending as far down as the periosteum of the bone. Although this fibrosis is a step in repair it may act as a deterrent to complete healing. When it occurs in the depths of the ulcer, the edges are tied down, and in addition, the fibrosis itself is apt to become hyalinized or calcified. (Fig. 3.) Accordingly, the varicose ulcer depends for its continuance not on the venous factor alone, but more particularly on the arterial and lymphatic as well. It should, therefore, be considered as a circulatory ulcer, rather than one merely due to varicose veins. These factors must be considered in therapy. If treatment is to succeed, it must be instituted and main-

tained before extensive fibrosis and scarring have occurred.

More than one etiologic factor may play a part in ulcer formation. For example, the simultaneous presence of varicosities and arteriosclerosis is common in our material; also syphilis with varicosities; and other combinations.

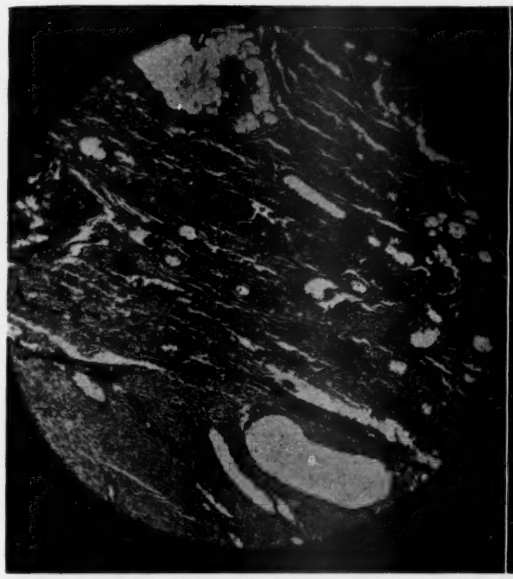

Fig. 2. Dilatation of venules; increase of tissue spaces; cellular accommodation; extensive deposition of pigment.

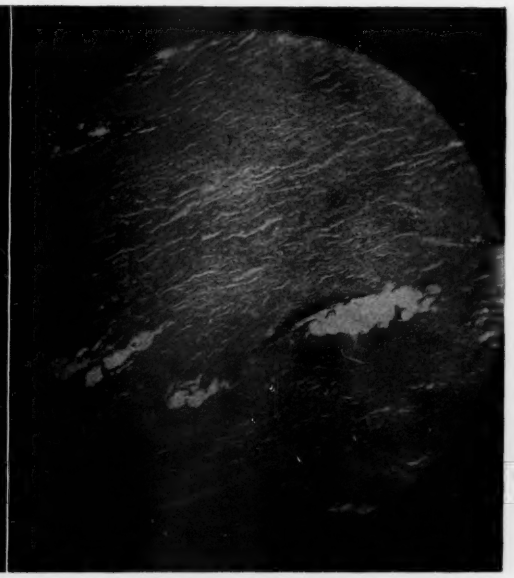

Fig. 3. Fibrosis and scarring in wall of chronic ulcer.

The diagnosis is not always easy. When varicose veins are the cause, we look for the following signs and symptoms—visibility of enlarged palpable veins which disappear or decrease in size on elevation of the extremity, edema on standing and the Trendelenburg, Perthés, and percussion tests.

The diagnosis of arteriosclerotic ulcer depends on the determination of the degree of arterial incompetency. The diagnostic methods include the Buerger postural test, pulses in the foot, color changes, symptoms of pain and claudication, *x*-ray to reveal calcification of the arterial walls, occasional arteriograms, routine oscillometry and temperature changes.

The luetic ulcer gives a definite history of infection and positive serology; the ulcer is covered with a firm grayish slough and the edges are raised and indurated, the margins punched-out in appearance. Such ulcers are often multiple and tend to be located chiefly on the outer aspect of the upper part of the leg.

The neoplastic ulcer may be primary or superimposed on the chronic varicose ulcer. We recently treated an unusual case of sarcomatous degeneration on a varicose ulcer. A thorough search of the world's literature revealed only two cases with this pathology. In malignancy, induration is always present and the appearance of the exuberant granulations is characteristic. As in the case of tuberculosis, biopsy establishes the diagnosis.

The tuberculous ulcer is commonly located in the vicinity of joints. It has a soft base and a grayish slough.

In all cases of leg ulcer a routine examination is made and a chart (Fig. 4) made up. In this way etiologic and contributory factors can be considered.

The basic principle underlying the treatment of leg ulcers is support to the circulation. In other words, we treat the circulation and not the ulcer directly. Local treatment is merely an adjuvant measure. The following therapeutic procedures have been used during the past ten years:

Local Measures. 10 per cent ichthyol; 1 per cent silver nitrate ointment; cod liver

oil ointment; balsam of Peru; zinc oxide; scarlet red; combined balsam of Peru, vaseline and lanolin; gentian violet; horse serum; aristol; insulin injected into margins of ulcer; histamine.

Results with gentian violet were poor, in that the ulcers were often aggravated. Scarlet red was found to be of value only when the ulcer was in the stage of beginning epithelialization; it was found best to alternate it every twenty-four hours with a mild ointment.

We came to the conclusion that none of these local measures which we had under observation had any special virtue and that they were of only minor value. Our experience with physiotherapy likewise showed that its value was doubtful. The methods employed were ultra-violet, natural exposure to the sun, infra-red, heat, diathermy, and iontophoresis. Incorporated in the latter was histamine and saline. The physiotherapy department experimented with copper iontophoresis with notably

PERIPHERAL VASCULAR CHART

NAME- OCCUPATION- NATIONALITY-
SEX- B.P.-
TOBACCO- WASS- ARTERIOSCLEROSIS-

TESTS:

1. LANDIS (HOT WATER IMMERSION 110°F-20 MINUTES).

	RIGHT FOOT	LEFT FOOT	ROOM TEMP.
CONTROL-	°F	°F	°F
HANDS -	°F	°F	°F

2. OSCILLOMETRY-
THIGH- UPPER 1/3 LEG- LOWER 1/3 LEG-
3. PULSATIONS-
DORSALIS PEDIS- POSTERIOR TIBIAL- POPLITEAL-
4. SKIN TEMPERATURE- BASE OF TOES-
5. POSTURE-
ELEVATION- PENDENCY- LEVEL-
6. HISTAMINE FLARE-

SYMPTOMS:

CLAUDICATION- BURNING SENSATION IN SOLE OF FOOT-
PAIN IN BUTTOCK- TROPHIC: NAILS-
REST PAIN- HAIR-
NUMBNESS- SKIN CHANGES-
WEAKNESS OF LIMB- MIGRATING PHLEBITIS-
ULCERATION- GANGRENE-
EDEMA-

VARICOSITIES:

LOCATION- EDEMA-
ULCERATION- TRENDELENBERG TEST-
TROPHIC CHANGES- PERTHÈS TEST-

FIG. 4.

poor results in that the ulcers enlarged in size and depth, with added increase in pain.

In those cases where the inflammation was acute, bed rest and wet applications gave relief. Hypodermic injections of histamine with the purpose of improving the peripheral arterial circulation were of questionable value. However, injections of acetylcholine hypodermically gave better results. Hypertonic intravenous saline, particularly in Buerger's disease and arteriosclerosis, was of definite value.

The operative procedures consisted of skin grafts, especially pinch grafts on ulcers covered with healthy granulations; vein sclerosing injections, ligation of veins, ulcer curettage, incision around the margins of the ulcer, biopsies and amputations.

Bacteriophage treatment consisted of an application of a water proof dressing which allowed the serum to accumulate.

The methods which gave actual results were the pressure bandage in various forms —ace bandage, Unna paste boot, elastoplast and adhesive. Pressure bandages were not disturbed for two or three weeks. This was a definite saving to the hospital because ordinarily it was necessary to change the dressings daily, and a saving in nursing time taken up by daily washing of dirty ulcers. The economy in avoiding innumerable dressings and bandages over a period of ten years was an economic side of the question which added up to many dollars and cents. Even in those cases where the odor of the ulcer secretions became objectionable, the pressure bandage was left intact. When necessary, deodorants such as powdered boric acid were applied over the pressure bandage itself.

The pressure bandage is placed over a small soft dressing which covers the ulcer. Thick dressings are avoided since they prevent even pressure. Equalized pressure accounts for a successful pressure bandage, otherwise more harm than good may result. When the secretion is profuse we use the ace bandage; when the ulcer discharge decreases the elastoplast and Unna paste boot are utilized. Before applying the pressure bandage the leg is elevated for one-half hour in order to reduce the edema.

Because of its paramount importance, it must be emphasized again that treatment must have as its prime objective the improvement of the circulation. Any local therapy is only of minor importance.

CONCLUSIONS

In the older age group both the venous and arterial systems of the lower extremities are involved. The compression or supportive bandage aids both systems—venous support relieves local anoxemia, etc. The local treatment of the ulcer is only of secondary importance except in those cases where other procedures are warranted by special indications.

CONCERNING THE SURGICAL PHYSIOLOGY OF DUODENAL ULCER

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THE pylorus forms the distal fixed extremity of the stomach. It lies a little to the right of the midline of the body, opposite the first lumbar vertebra, with the pancreas below and behind and the left lobe of the liver above and in front. The cardia forms the proximal fixed extremity and between these two points the stomach is suspended from the gastrohepatic ligament. The cardiac sphincter guards the entrance and the pyloric sphincter the exit, the former being related to the motor activity of the esophagus and the latter to that of the stomach. The pyloric sphincter is a definite anatomic structure, the best developed of all the sphincters of the gastrointestinal tract, being represented by a local increment of circular muscle fibers about ¼ to ½ inch in width. During operations under general anesthesia the sphincter is relaxed, making definition difficult, but its location is readily marked by the transverse pyloric veins.

The arteries of the stomach do not reach the pylorus; the latter is supplied from above by the pyloric artery, a branch of the hepatic from which are also derived the branches supplying the first portion of the duodenum.

The anterior and posterior branches of the vagus nerve supply the respective aspects of the stomach but only as far as the pylorus. The latter structure is innervated from above by fibers of the hepatic branch of the vagus which probably also carry sympathetic filaments.

Since the nerve supply of the pyloric sphincter has been the subject of much controversy it may be well to deal with it more fully. Embryologic and comparative anatomic studies cited by Gaskell show that the sphincters of the gastrointestinal tract have developed from the dermal musculature and at one time formed a continuous muscular investment of the gut. In the course of development most of the dermal musculature disappeared from the gut and that which remained became localized at certain points to form the sphincters.

It is well established that the sympathetic nerves which represent the thoracicolumbar outflow of the involuntary nervous system supply motor fibers to all blood vessels and sweat glands, to all unstriped muscle under the skin and to tubular structures derived from the segmented duct. Structures with a common origin usually have a common nerve supply. It has been proved that the motor nerve for the ileocecal sphincter is from the superior mesenteric ganglion. The pyloric sphincter in all likelihood belongs to this group and is supplied by motor nerves from the sympathetic. The vagus, a parasympathetic nerve, supplies inhibitory and dilator fibers to this sphincter and has its ganglion cells located in the wall of the gut. Experimental attempts to localize the nerve supply of the pyloric sphincter have shown conflicting results, probably because of failure to differentiate between sphincter and antral activity as related to the emptying of the stomach, and because of the difficulty of separating vagus from sympathetic fibers. The vagus is not only the motor nerve for the stomach but it is also the inhibitor and dilator nerve for the pyloric sphincter. Cutting it does not disturb the intrinsic mechanism of the stomach which is controlled by local ganglonic cells. The motor activity of the sphincter is under sympathetic control which is nicely correlated with the motor function of the stomach and is also ex-

tremely sensitive to extraneous stimuli not only from the abdomen but from the entire body.

Horton showed by anatomic studies that the musculature of the pyloric sphincter is made up principally of circular fibers whose contraction closes the orifice. He demonstrated also that looped around the circular fibers are muscle bundles prolonged from the longitudinal muscle coat of the stomach. Contraction of these latter fibers which are under vagus control serves to dilate the sphincteric orifice and to hold it open. Thus it becomes apparent that the mechanism of the pyloric sphincter resembles that of the iris of the eye.

The normal function and behavior of the pyloric sphincter have to do with regulating the passage of food from the stomach to the duodenum and probably with the regurgitation of duodenal contents into the stomach. Recent investigations indicate that acidity on the gastric or duodenal side of the pylorus does not normally control the activity of the sphincter. Each advancing peristaltic wave in conjunction with increased tonus of the stomach overcomes the tonus of the pylorus and forces some of the chyme into the duodenum regardless of whether the reaction of the chyme is neutral, acid or alkaline. The pyloric tone normally only keeps the sphincter closed when the pressure on either side is insufficient to overcome it.

Structures which are controlled by the sympathetic nervous system exhibit reactions which are peculiarly responsive to many stimuli. Perspiration and pallor of the skin, involuntary evacuation of the bladder and rectum induced by fright and emotional shock; hyperpiesis resulting from worry and nervous strain and from trauma to an extremity may be cited as results of overstimulation of the sympathetic nervous system. Indeed many of the symptoms of surgical shock may be explained in a similar manner. Postoperative intestinal ileus is not caused by paralysis of the intestine, but is due to a preponderance of sympathetic activity, causing inhibition of peristalsis and spasm of the sphincters. Spinal anesthesia or severence of the sympathetic nerves promptly relieves or prevents this condition. Mere sudden forcible contraction of the stomach would cause ejection of its contents into the duodenum; therefore, for vomiting to occur there must be coincident closure of the pylorus by contraction of its sphincter and reversal of gastric peristalsis.

All stimuli which affect the sympathetic nervous system do so by causing an increase in the output of the adrenal glands. The action of epinephrine is limited to structures having sympathetic innervation, there being no effect on nerves of the autonomic system. The sphincters are made to contract by sympathetic stimulation and their tone is probably increased by atropine which paralyzes the local inhibitory ganglia.

By application of this general knowledge of sphincters to the pyloric sphincter it is presumably correct to say that normal tonus maintains closure of the pylorus, that it relaxes and opens in response to vagus stimulation as it occurs in gastric periastalsis, and that spasm of the pyloric sphincter occurs from local or reflex irritation.

Surgical diagnosis must frequently differentiate between gastric symptoms caused by intrinsic disease of the stomach and gastric symptoms which are manifestations of extragastric lesions. Ulcer of the stomach or duodenum is a local irritative lesion whose symptoms are directly related to the functional activity of the stomach. The regularity and periodicity of symptoms, especially of duodenal ulcer, are constant from day to day. The stomach often exhibits hypermotility and there is usually spasm or achalasia of the pyloric sphincter. The etiology of duodenal ulcer is as vague as ever. Its experimental reproduction has not simulated the conditions found in the human. Infection seems to play a part but the predisposing factors, especially those which promote chronicity, remain unknown. The fact that duodenal ulcer occurs so often in certain

individuals of a characteristic habitus described by Draper and Hurst, that it is often found in several members of a family of the same and succeeding generations, that it is often prone to recur after surgical treatment, that it seems to bear some relationship to the use of tobacco (nicotine paralyzes the vagus terminals), and that the symptoms of ulcer are often accentuated by worry or nervous strain are highly significant and suggestive of a nervous influence in etiology.

Reflex spasm of the pylorus may be caused by any abdominal lesion which irritates the peritoneum. It is often a sequel of abdominal operations, and may even arise from the ligation of the sac of an inguinal hernia done under local anesthesia. Spasm from the above causes is usually temporary, sometimes recurrent at irregular intervals and rarely of much significance. Continual clonic spasm produces hypertrophy of the sphincter as seen in early infancy. The etiology of this condition seems to be best explained by overstimulation of the sympathetic fibers to the sphincter. It has been suggested that hyperadrenalism causes the overstimulation. Mere cutting of the sphincter, however, suffices for cure.

Reflex spasm of the pylorus in the absence of duodenal ulcer and other abdominal lesions may give rise to symptoms which often simulate duodenal ulcer and justify operation. Bastianelli speaks of such cases, which he has treated successfully by the Ramstedt operation. Gastroenterostomy in the absence of ulcer does not relieve the symptoms of pylorospasm, but may cause additional symptoms which are only relieved by undoing the anastomosis.

The significant features of duodenal ulcer which may be explained by the activity of the pyloric sphincter are the chronicity and periodicity of symptoms; the unchanging cycle of pain-food-ease-pain; the hypermotility and hyperacidity of the stomach; the selective location of the ulcer in the anterior superior wall of the duodenal cap about 1 inch from the pylorus; the absence of symptoms for weeks or months when an unhealed ulcer is known to be present and the occasional occurrence of ulcer symptoms in the absence of ulcer. Certainly the supposition is justified that the behavior of the pyloric sphincter has much to do with the symptoms of ulcer. Is it possible that misbehavior of the sphincter from direct or reflex nervous influence is a factor in the production of duodenal ulcer?

Mere excision of duodenal ulcer has not been followed by satisfactory results, and various types of plastic operations on the pylorus have yielded a large percentage of failures. The reason for failure in the latter may be that the sphincter is rendered only temporarily inactive because subsequent operations have often shown little change in the appearance and caliber of the pylorus. The procedure which Judd has used with eminently satisfactory results in a large number of cases is excision of the ulcer combined with removal of the anterior half of the pyloric sphincter. By this operation sphincteric activity is more or less permanently abolished. There can be no doubt that the removal of a large part of the pyrloric sphincter is the major factor in the good results which follow this operation. Excision of the anterior half of the sphincter probably removes many of the local ganglionic cells and the attachments of many sympathetic fibers. It is possible that after a time the sphincter may heal and resume activity. In this event, if certain predisposing factors persist, there may be recurrence of duodenal ulceration. The solution of the permanent control of duodenal ulcer remains involved with its etiology in which dysfunction of the pyloric sphincter resulting from reflex nervous influence should seriously be considered.

Hyperacidity is the most constant laboratory finding in cases of duodenal ulcer. Just what is hyperacidity? Does it mean a larger amount of acid, or acid that is stronger than normal or both? Certain basic facts of the chemistry of the stomach must be kept in mind. The acidity is due entirely to hydrochloric acid. Pavlov showed that the gastric juice as it flows

from the glands possesses a constant acidity—0.5 per cent—never more, never less. The optimum acidity for digestive purposes is 0.2 per cent. The reduction is accomplished by regulation of the amount of acid secreted, by dilution with food and fluid entering the stomach, by neutralization from any alkali in food, fluid, and stomach mucus, and at special times by regurgitation of duodenal contents into the stomach. The acid-secreting mechanism maintains the optimum level of acidity when there is digestive work to be done, and as Pavlov says, the exactitude of the work of the glands is astonishing; that which is demanded of them they furnish each time to a hair's breadth, no more and no less.

Surgical duodenal drainage, evulsion of pancreatic ducts, and formation of a pancreatic fistula are among the most successful experimental methods for the reproduction of peptic ulcer. In these experiments it is a significant fact that one starts with a normal gastric function. Later, in spite of the fact that an ulcer has been produced just beyond the outlet of the stomach, and that all likelihood of duodenal regurgitation has been eliminated, nevertheless gastric function, especially the behavior of the acidity remains unchanged.

In view of these findings can we maintain that ulcer causes hyperacidity? Can there be any doubt that the normal stomach regulates its own acidity? I am aware of no experimental method which has been successful in producing a maintained hyperacidity. From the practical standpoint, hyperacidity means not stronger acid, but the secreting of normal acid beyond the quantity required for digestion and probably when there is no digestive work to be done. Hyperacidity then is really hypersecretion.

Where does the fault lie? Not in the stomach itself. It seems very probable that this dysfunction of gastric secretion originates in the central nervous system, and for reasons not necessary to explain here, it is part of a fixed constitutional pattern. The psychic phase of gastric secretion produces the most potent juice; it anticipates food in the stomach. "Appetite spells gastric juice." I believe that in the patient with duodenal ulcer the psychic phase of gastric secretion is not only overactive but active at unnatural times. The result in the ulcer patient is a constant effort, conscious or unconscious, to satisfy and neutralize the excessive acidity. Hence the increased appetite, the comfort from food in the stomach and the solace from alkaline powders.

Why do not all persons with hyperacidity have ulcer? Is the ulcer the cause of so-called hyperacidity? Is it responsible for achalasia and spasm of the pyloric sphincter? The symptomatology of duodenal ulcer is explained by hyperacidity and achalasia or spasm of the pyloric sphincter and not by ulcer. Ulcer is the result of the above and not the cause. Removal of the ulcer does not affect hyperacidity nor does it relieve the symptoms, but the latter are cured in many instances by gastroenterostomy even though the ulcer remains for a varying period of time until healing occurs. In the remission periods the patient with duodenal ulcer still has his ulcer although it is probably in a healing stage.

It is not uncommon to see a patient who has the typical symptoms of duodenal ulcer but no ulcer. Reflex spasm of the pyloric sphincter has been known to surgeons for many years. Moynihan painted its word picture in cases of chronic appendicitis. Bastianelli cut the sphincter and relieved patients who had the symptoms of ulcer without actual ulcer. If these patients had been allowed to go on without treatment, would they have developed ulcer? Pylorospasm gives rise to local pain and discomfort, but not to hyperacidity. Spasm of the sphincter is a reflex sympathetic nervous mechanism. Its cause may be any peritoneal irritation, any mechanical derangement, such as partial obstruction, an adherent appendix, an omental attachment to an incisional scar. Food allergy may be a factor—there

is always spasm of the sphincter during an attack of migraine.

Out of all the experimental work in the search for the cause of chronic duodenal ulcer one fact stands unshaken, that is, that the hydrochloric acid of the stomach has a great influence and is probably the single direct cause of ulcer in the human being. The mechanism and factors by which this is brought about amount essentially to this: hypersecretion of hydrochloric acid, due to a constitutional fault, is combined with failure or inadequacy of the normal safety controls (stomach mucus, alkaline pyloric secretion, and duodenal regurgitation, which carries the strongest alkaline secretion in the body). There is ample evidence of duodenal regurgitation in the normal person, i.e., the frequent finding of bile in the stomach; reverse duodenal peristalsis, demonstrated by the *x*-ray; and the intractable nature of a postoperative duodenal fistula. Interference with duodenal regurgitation depends primarily upon the pyloric sphincter, either its failure to relax, known as achalasia, or actual spasm. The latter has been frequently demonstrated in patients with duodenal ulcer.

The surgeon does not have to seek outside his clinical experience for a demonstration of the experimental production of peptic ulcer. Every gastrojejunal ulcer is an example of experimental peptic ulcer in the human being.

There have been many types of operations used in the treatment of duodenal ulcer. Their success depends essentially on the control of the hypersecretion of hydrochloric acid. In the evaluation of postoperative results it is important to remember that the patient continues to have hypersecretion although his symptoms may be relieved and his ulcer healed. Simple excision of the duodenal ulcer has been abandoned because of the high rate of recurrence and mere closure of a perforated ulcer is followed by unsatisfactory permanent results in about 35 per cent of cases. Evidently the ulcer alone does not warrant therapeutic attack.

Plastic procedures designed to enlarge the pyloric-duodenal avenue, when well done, have been followed by remarkably few recurrences of ulcer. Their failures have been due to mechanical interference with the emptying of the stomach caused by extensive local adhesions which so often follow these procedures.

Gastroenterostomy almost invariably brings about healing of the original ulcer in the duodenum, but in a varying percentage of cases a new ulcer forms in the jejunum near the anastomotic stoma. We have learned to know that a person with a gastroenterostomy is never free of the potential danger of a marginal ulcer. I have notes of a patient who was under my care for this condition twenty-eight years after a gastroenterostomy had been done for duodenal ulcer. The answer to the failures after gastroenterostomy may be found in the explanation of its successes. I have no doubt that the successful surgical treatment of duodenal ulcer by any type of operation depends upon efficient control of hyperacidity. The test of this does not lie in the results of gastric analysis but in the continued well-being of the patient. No operation yet devised can alter the cerebral stimulus upon which hyperacidity depends.

It is a human failing to carry on to extremes, even in therapeutics. An example of this is the practice of partial gastrectomy for duodenal ulcer. The stated reasons have to do with removal of the ulcer-bearing area of the stomach and duodenum and its associated gastritis, and with a supposed control of hyperacidity by removal of part of the secretory area of the stomach. Gastritis has nothing to do with the cause of ulcer although the two may have a common origin. Removal of part of the secretory area does not slow up the activity of the part that remains, which actually may undergo functional hypertrophy. Marginal ulcer may occur after partial gastrectomy for duodenal ulcer and its incidence is almost the same as when gastroenterostomy has been done in well selected cases. If anything can be said in justification for gastrectomy for ulcer in

the duodenum, it is that its mechanics insure more adequate neutralization of gastric acidity. But the sacrifice of three-fourths of the stomach is a big price to pay for the removal of a small ulcer in the duodenum.

Assuming that the background of duodenal ulcer with its associated perversion of gastric physiology is of the nature depicted elsewhere in this paper, what is a logical solution to the problem? If hyperacidity and dysfunction of the pyloric sphincter are at fault, is there any method to correct them? So far there is no mechanical means to attack the basic fault of hyperacidity. In duodenal regurgitation nature has its own method of control, but in the ulcer patient its action is prevented by the pyloric sphincter. This is not a new thought—surgeons long ago tried to render the sphincter inactive by cutting its fibers. The results were immediately good, but were only temporary, because after healing the sphincter resumed its former activity. The method of Judd by which the anterior half of the sphincter is removed comes very near the ideal procedure. Its success in cases in which it can be done far surpasses any other operation for ulcer. Its small percentage of failures is due not to recurrence of ulcer but to mechanical factors incident to local adhesions. Judd's operation opens the stomach and duodenum and is completed as a gastroduodenostomy. It is liable to the same faults due to adhesions which have followed other plastic procedures on the pylorus.

The ideal method to obtain our objective is removal of the anterior half of the pyloric sphincter without opening the stomach or duodenum. This we have done by a method described elsewhere, with excellent results. The sphincter has been rendered permanently inactive or at least no longer interferes with duodenal regurgitation. The operation is suitable only for those duodenal ulcers in which extensive scar tissue and fibrosis are absent. Otherwise identity and removal of the anterior half of the sphincter are not practical.

SUMMARY

The pyloric sphincter is composed of constrictor and dilator muscle fibers, the former under the control of the sympathetic nerves and the latter under the parasympathetic or vagus nerves. The sphincter plays a part in the emptying of the stomach and also in the regurgitation of duodenal contents into the stomach. Its mechanism may be disturbed by local or reflex irritation. Many of the clinical features of duodenal ulcer can be explained by the activity of the pyloric sphincter. It may cause the typical symptoms of ulcer when no ulcer is present, since the ulcer itself does not cause the symptoms which identify its presence.

Dysfunction of the pyloric sphincter is one of the two main factors in the etiology of duodenal ulcer. The other factor is hyperacidity. The normal stomach regulates its own acidity. In certain individuals hypersecretion is a fixed constitutional fault and its control rests on the mechanism of duodenal regurgitation. Failure of this control means the development and maintenance of a duodenal ulcer. The pyloric sphincter because of spasm or failure to relax becomes a potent hindrance to the regurgitation of alkaline duodenal contents into the stomach.

Successful treatment of duodenal ulcer depends on the control of hypersecretion. The administration of antacids and the use of sedative measures to slow up the cerebral stimulus constitute the accepted medical treatment, but failure is common. Surgical measures have been designed to utilize the patient's own control mechanism. Failure means the development of a new ulcer. Of the two factors concerned in the etiology and maintenance of duodenal ulcer, dysfunction of the pyloric sphincter alone is amenable to direct attack. Its function can be abolished permanently by a simple operation which removes the anterior half of the sphincter without opening into the duodenum or stomach. This procedure has been carried out with enduring success in cases of duodenal ulcer.

EXPLORATORY LAPAROTOMY

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THE title of this article is self-explanatory—a laparotomy undertaken before a diagnosis has been made. With our modern methods of diagnosis it is perhaps true that the need for the performance of such an operation is less frequent than formerly. Still we are forced, much to our chagrin, to admit that today, even in the best clinics of the world, exploratory laparotomy is far from being an infrequent operation. And it is highly probable that it is of greater frequency in the better clinics for the simple reason that the cases in which it comes to be indicated, particularly those wherein it is not immediately indicated, are likely to be referred in greater numbers to the greater clinics. The indications for an exploratory laparotomy may vary a good deal in various hospitals, and "they vary with the skill, qualifications, temperament and conscience of the surgeon in charge of the case" (Schnitzler). But there *are* clear-cut indications for the performance of such an operation and also certain contraindications.

An exploration of the abdomen by surgical operation is indicated for therapeutic purpose in any chronic *intra-abdominal* condition thought to be relievable by surgical means, but the exact diagnosis of which has not been arrived at after the exhaustion of all the skill and methods that medicine has to offer. It is also indicated in any acute abdominal case, thought to be surgical, wherein the time lost in waiting for the performance of the various diagnostic tests and manipulations in ordinary use, or where the very tests and manipulations themselves might increase the risk to the patient's life. Also in certain cases in which the diagnosis has already been established (e.g., cancer of the rectum, etc.) but where the extent of involvement is unknown and hence the proper form of therapy cannot otherwise be agreed upon.

It is strongly contraindicated when the surgeon has reason to feel that the operation, while it may make the diagnosis positive, yet cannot hope to give relief. Never should an exploratory laparotomy be done for biopsy only; and it is also strongly contraindicated when there is reasonable certainty that the condition of the patient will be made worse thereby.

Attempts have been made in the past, and let us hope will continue to be made in the future, to discover some means to enable us at least to arrive at a diagnosis, and, if possible, administer therapy in some other way than by recourse to a major operation. The invention of the cystoscope by Nitze in 1876 has, I believe, been responsible for saving and prolonging more lives than were lost altogether by war and pestilence ever since. Kelling, in 1901, applied the same instrument—modified—to intra-abdominal diagnosis and since then, from time to time, in various countries reports of improvements of technique and instrument have been brought to our notice. But as yet there is no place where its use has become so accurate as to displace exploratory laparotomy. The gastroscope is at present exciting about as much attention as the gastrodiaphane did some thirty or forty years ago (which is not saying a great deal), but the proctoscope and sigmoidoscope are second only to the cystoscope for the illumination of hitherto dark areas in the field of diagnosis. There is no doubt, however, that these means, and particularly the *x*-ray with the barium meal or enema and later pneumoperitoneum and the Graham gall-bladder test

have been responsible for the tremendous advance that has been made in abdominal diagnosis in the past forty years.

It has been clearly stated that exploratory operation is indicated only in *intra-abdominal* disease or injury. We shall consider the acute conditions first because of the great importance in these of an early answer to the question of diagnosis. Let us keep in mind the fact that there are pathologic conditions elsewhere that may betray their presence only by intra-abdominal symptoms. The chest is often the abode of such processes. Who has not seen the early pneumonia, particularly in the child, mistaken for acute appendicitis? The key to the differentiation is in the careful examination. The high fever, increased respiration, the very high white count, the absence of deep tenderness, and the presence of chest findings are most unusual in acute appendicitis, while nausea, rigid rectus and tenderness, particularly on deep pressure are very common accompaniments of acute appendicitis. A deep inspiration can hardly be made at all by one with acutely inflamed lung or pleura and its forced attempt fails in a grunt or slight cough, whereas in acute appendicitis only the *lower* abdomen remains quiet with deep inspiration—no cough and no increased pain are present. Particularly in children, there is the acute peritonitis that accompanies "flu" or follows tonsillitis. This should remain a medical case throughout. An operation is altogether too frequently followed by a funeral.

Most dramatic and most terrible is the resemblance between the acute abdominal catastrophe and the acute cardiac crisis. Angina pectoris, coronary thrombosis—the very names strike a chill to the heart of every physician over fifty, and every medical student early becomes familiar with their history, symptoms and sequentia. Can it be possible that an exploratory laparotomy could ever be performed in these conditions or in that of acute cardiac dilatation? It surely has been done and in all probability will be again. There are many cases cited in the literature. Here is one from St. Mary's hospital:

Case I. C. V., male, 42 years old, a salesman, was carried in complaining of terrific abdominal pain, worse in the epigastrium, which had begun three hours before, just after lunch. Nausea and vomiting were present. He was of stocky build, not fat, and had always been healthy and industrious. He had eaten a hearty lunch and had got into his auto to go to work. He felt nauseated and opened the door, then remembered no more till he was being lifted from the street beside his car, when he began to scream with the pain in his abdomen. A physician gave ¼ gr. of morphine by hypo and after half an hour repeated it because of the terrible pain.

The abdomen was rigid, the muscles tense, and the nausea persisted. W.B.C. numbered 17,000 (infectious picture); temperature was 100.6, pulse 76, and blood pressure 110/85. There was not the expected tenderness. An *x*-ray revealed no free gas and operation was considered inadvisable. A nasal tube was inserted and the stomach kept empty.

Next morning he was symptom-free but a little nervous. The third morning he was hungry and thirsty, but the temperature was 101.8, pulse 132, respiration 24, and a mass was present in epigastrium and right upper quadrant. The abdomen was distended and very tender in that area, and the white blood cells were 17,700.

The patient was taken to the operating room. After failure of spinal anesthesia, ether was used. He became cyanosed and remained so. The abdomen was opened. The liver was enormous and purple, the gall-bladder, pylorus and duodenum were normal. A cystic mass, 2 inches in diameter, of robin's egg blue was seen behind the peritoneum to the right of the duodenum and in the edge of the gastrohepatic omentum. Aspiration drew dark blood from it but did not diminish its size (the portal vein or the vena cava?)! The wound was closed, and the patient died within the hour. The post-mortem showed coronary thrombosis with dilated heart.

And just to show the converse, the following is cited:

Case II. A. F., male, 54 years old, a real estate man, known for years to have duodenal

ulcer, at last decided to be operated upon. About seven o'clock of the evening before operation he was suddenly stricken with terrific pain in the left side of the chest, neck and left arm and leg, and at once went into shock with copious sweating. The abdomen was neither tender nor rigid. The handling of it annoyed him and he begged to be let alone. He lay quietly and constantly on his right side with abdomen slightly turned to the bed. The diagnosis was angina pectoris. We didn't know enough then of the value of an *x*-ray plate. He died in eight hours and the post-mortem revealed a perforation 2 cm. in diameter in a duodenal ulcer!

These are hard cases to diagnose because the symptoms all seem to indicate with unerring certainty the site of the disease. But even in the most clear-cut case we ought to think of the possibility of error. There are two aids now nearly always available. The electrocardiogram is pretty certain to reveal any serious cardiac disease and the *x*-ray, showing free gas in the cavity, says positively that the gastrointestinal tube has been ruptured. Of course, there is not always gas, but when there isn't, the perforation is not leaking very much. It has been said that the pallor of the acute cardiac crisis contrasts as a rule with the ashen cyanosis of acute pancreatitis. It is often true, but the cases cited above show that it is not always so. And while most exquisite tenderness in the region of the liver, together with or preceding its rapid enlargement, is very common in acute cardiac dilatation, yet this tenderness is uniform even to the right lower margin, but in the upper abdominal catastrophe the tenderness is always greatest over the site of the disease and spreads from there as the hours pass.

Among rare conditions one must think of are angioneurotic oedema or purpura with gastrointestinal lesions. There is nausea and always severe, cramp-like pain and, maybe, rigidity of the abdomen. The intestine is usually active and borborygmi can be heard—and presently the enema brings bloody stool. This is not easy to differentiate, but the short clotting time and long bleeding time might make us at least think of purpura.

An acute abdominal crisis often follows within an hour after the "black widow" spider bite. The symptoms have led to laparotomy but even though nausea, vomiting or cramp-like pain is present, with muscular hypertension, one should notice that the rigidity is not confined to the abdominal muscles.

An exploratory laparotomy for vomiting alone should never be undertaken until all toxic causes, not forgetting uremia, and, in the female, pregnancy, have been ruled out. Just to think of the possibility will positively prevent one from making the mistake. But vomiting alone may be the only symptom in the patient with a high obstruction or an internal strangulation of omentum, or one which does not include the whole gut.

There are diseases of the nervous system which give rise to crises of severe abdominal pain, and even to abdominal pathology. All are familiar with the fact that there are gastric crises in tabes dorsalis and other spinal cord diseases, but not so many know that the abdomen has been opened and appendix or gall-bladder removed for crises of abdominal pain caused by a brain tumor! Indeed it is true that pathology in, or operations on certain parts of the brain have been the cause of perforating ulcers in stomach, duodenum or large bowel. The latter I have had in my own practice. And for the upper parts of the tract there are many cases cited by Cushing and others. I have seen acute spinal meningitis very closely simulate an acute abdominal inflammation. It was the hyperesthetic skin of legs and feet and the lack of tenderness (deep) that made us look for other signs and prevented an immediate laparotomy. To think of the possibility enables us to avoid any mistake, for these diseases of the central nervous system usually have other characteristic signs and symptoms.

Acidosis preceding diabetic coma has been accompanied by abdominal symp-

toms and signs suggesting acute catastrophe, and the abdomen has been opened to find no pathology present. Fever may be present, with a high white count; in fact, the white count has been as high as 50,000, and has been accompanied by nausea, vomiting and pain. A urinalysis nearly always reveals the truth, but the blood sugar never fails to do so. The unfortunate fact remains that diabetics do sometimes have real pathology in their abdomens.

A crisis due to lead colic will not lead us astray. The low white count, low temperature, slow pulse, the blue line in the gums and stippled red cells and the rolling and writhing about of the patient, consign him to the care of the physician.

Very often indeed an operation for an acute abdominal catastrophe must be an exploratory laparotomy. The thing to be certain of is that its pathology is intra-abdominal. The case is so urgent, since, if relief is to be of value, it must be given at the earliest possible time after the onset of the disease. To lose valuable time in making tests and even the very procedure of making the tests, e.g., *x*-ray, gastrointestinal examination, etc.—may lessen the chance of saving the life of the patient. While it is desirable to make certain of the diagnosis before operation, to take the time and use the means necessary to enable us to do so might allow the disease or condition to progress to such a point that no surgeon in the world could save that patient's life by operation. We must still be strong in the opinion, "It is better to look and see than to wait and see." It is much better to operate without a diagnosis and prevent death than to wait for the correct diagnosis and have a funeral. After all, it does not make much difference to the surgeon whether it be a gangrenous gut or gall-bladder, a perforated ulcer or an acute appendix, a cyst with a twisted pedicle or an acute pancreatitis. But there are some conditions that he must make sure it is not. Of some of these we have spoken, and there are three or four more which may lead to an operation followed by regret or even remorse.

The stone in the ureter and the acute pyosalpinx or the parametritis following abortion are dangerous pitfalls. The urologist can now exclude the former very definitely. The radiation of the pain, the aching in the loin and the complaint of a sense of fatigue should serve to put us on guard against kidney or ureter. The parauterine infections have the habit of starting off with chill and high fever, and the pelvic examination usually decides the diagnosis and defers the operation. An acute diverticulitis of the sigmoid should be excluded before one decides to explore an acute case. The location, the pain and soreness, the preceding constipation and the fever should aid us. The *x*-ray with barium enema, taken in the acute stage, is likely to be reported as "a filling defect," but carcinoma there doesn't present the picture of an acute abdomen.

In these acute abdominal crises, one must operate without waiting for the certain diagnosis. Only be certain that it is intra-abdominal and that it is a surgical condition. There is no other situation in his life which tests the surgeon like one of these: if he operates and fails to find pathology he loses his reputation, but if pathology be present and he doesn't operate the patient may lose his life. And when he fails to find pathology and the patient loses his life as a consequence of the operation, that is worst of all. If he waits to make certain of his diagnosis he will unfortunately most likely have it confirmed at the post-mortem —rather poor satisfaction.

So much for the acute abdominal crisis of intraabdominal origin. I have purposely refrained from rehearsing the signs and symptoms. The operation gives the best hope of saving life in all of these acute conditions, and usually the earlier the better—usually, but not always. Not while the patient is in shock and untreated for it. Naturally, I am not including acute intestinal obstruction among the conditions in which exploratory laparotomy is indicated,

as it should be, and usually is, easily diagnosed before operation.

There are some conditions in which the indication for exploratory laparotomy may be debated—acute hematemesis of unknown origin is one, and suspected subdiaphragmatic suppuration is another. In the first, one must remember that the cause may be unrelievable by surgical operation and that when the cause cannot be found with our present methods—that is to say, when the diagnosis cannot be made—the case is best treated medically. In the subdiaphragmatic suppuration the outlook under medical care is so bad that the patient has more to hope for from proper exploration.

The exploratory laparotomy is indicated in certain intra-abdominal injuries. There may exist a wound of the parietes or not. Suppose that not even a contusion can be found, but there is a history of recent injury and the patient presents signs of intra-peritoneal irritation or of loss of blood and is getting worse. I feel sure one should not wait longer than five or six hours before exploring. There is, I think, no viscus within the abdomen or pelvis that has not been accidentally ruptured without a surface sign of injury. But the history is always there and the symptoms appear in time to show the danger, but they may not be perceived. The abdominal muscles become rigid, peristalsis slows down or ceases entirely in the region of foreign matter within the cavity, pain and tenderness come later and usually still later the nausea and vomiting. Has the urine been examined for blood? Has the bladder capacity been tested to determine that its walls are still intact? When the kidney alone is ruptured without tear of the peritoneum the signs of peritonitis do not supervene, but when the gastrointestinal tube is broken the *x*-ray will soon reveal free gas.

In all cases of gunshot, stab, or other wounds of the abdominal wall, or in cases of such wounds of near lying parts of the body, if one has reason to suspect that the abdominal cavity has been penetrated, there is no argument about it: the abdomen should be opened and explored just as soon as possible. In such early cases, shock, if present, is likely to be increased by, if not due entirely to, the loss of blood. Therefore, a transfusion and an exploratory laparotomy should be begun without loss of time.

There are also chronic conditions arising within the abdomen wherein the physician, after exhausting all the means of making a diagnosis, is unable to say positively just what the condition is. He may fear that while he is waiting for the advent of such signs or symptoms as will enable him to be assured, the disease may make such progress as to place the patient beyond the hope of help. In all such instances, exploratory laparotomy is indicated.

Here I would warn all, but especially the young surgeon, against that class of patients—and they are very numerous—who speak so convincingly of their terrible sufferings (Dr. C. H. Mayo said "They enjoy poor health, their folks suffer."), that one might be forgiven for making a diagnosis of chronic appendicitis or of almost anything very serious. They have symptoms, perhaps signs too, but usually the most painstaking and complete search at laparotomy fails to reveal pathology. "Methinks he (or she) doth protest too much" to have need for exploration. The first operation on the neurotic is one of a series and very rarely indeed is pathology found to explain the symptoms. When one has failed to find pathology he has done an unnecessary operation.

There are some cases of chronic abdominal pain that have ample reason for being and they may perhaps be relieved by an exploratory operation. In all such, we must be certain that disease of the cord or of its membranes, of the spine or ribs and of the kidneys are excluded. Inflammations usually give more than pain, but often neoplasms do not give much else.

Then there are conditions wherein exploratory laparotomy is indicated but the fact is not yet generally agreed upon. Let

me give special consideration to cancer of the stomach. The popular operation for cancer of the stomach in our country today is simple gastroenterostomy. Numerous patients who have had gastric resections live for five years or more, but it must be borne in mind that these are *carefully selected cases*. However, the bulletins of the clinical society, listing the work for the day, show very few operations for cancer of the stomach, and of these, the scheduled gastrectomy often turns out to be a simple gastroenterostomy. I have seen only a few cases of cancer of the stomach in which the diagnosis was made before operation, in which the disease had not already progressed beyond the point of possible cure. This being so, we must admit that its diagnosis is not being made early enough.

In order to help to save more lives from this disease, let me say here that every case of suddenly appearing stomach trouble in a man (or woman) over forty-five which doesn't respond to proper medical care within a period of four weeks, should be subjected to exploratory laparotomy. We should not wait longer for the appearance of physical signs—the *x*-ray reveals the cancer only when it is larger. Unfortunately, we wait for the report containing those most sinister words, "There is a filling defect." This short sentence is usually a sentence of death! In the present condition of our ignorance, early exploratory laparotomy for all stomach cancer suspects will do for cancer of the stomach what has been done for breast cancer. And we must strive to make this the practice. The person in the cancer age who has all his life been free from stomach trouble and for the first time begins to have serious stomach complaint, is always a cancer suspect, and as soon as we come to realize this the prognosis in cancer of the stomach will be better.

In certain cases of chronic jaundice an operation is indicated for both diagnosis and treatment—in certain others for treatment alone. It has been said that painless jaundice has malignant disease for its cause, but this is not always true. Furthermore, there is no known test or method of examination whereby one may, in the absence of palpable tumor, be assured of the existence of cancer of the papilla or of the pancreas or of the absence of a stone. If one does not know the cause of the jaundice, but one is certain that it is of the obstructive type, that is sufficient reason for exploratory laparotomy (providing the patient is not hopelessly ill). Many such obstructions have I found due to removable and curable strictures and stones, and some supposed even at operation to be cancers of the pancreas have been cured by anastomosing the gall-bladder to duodenum or stomach. That is to say, cancer of the pancreas was not present at all, as the following case will show:

Case III. S. T., male, 54 years old, a lawyer, tall, well built, who weighed 200 pounds but whose best weight was 230 pounds, complained of nausea and vomiting and dull, aching pain in the right upper quadrant. His appetite, usually good, had become poor. His bowels, previously constipated, were now diarrheic, the stool grayish and very foul. Urination was normal. General examination proved negative, except for slight tenderness in the epigastrium to the right of the midline. R.B.C. were 5,320,000, W.B.C. 5,200. Hemoglobin was 80 per cent, clotting time three and three-quarter minutes, blood pressure 142/88, N.P.N. 43, blood sugar 125, Wassermann negative, icterus index 23, Graham test negative. The urine was acid with specific gravity of 1.016; albumin plus; a trace of sugar; some red and white cells, but no casts.

Diagnosis could be chronic pancreatitis, chronic hepatitis or cancer of pancreas.

An exploratory laparotomy was done. The liver was seen to be somewhat shrunken with thick rounded edges and thickened capsule. The gall-bladder contained black bile, with no concretions or adhesions. The stomach was free, with neither scar nor masses. The pylorus was smooth and the duodenum was wider than normal and seemed dilated. The head of the pancreas was very hard and somewhat larger than normal. The diagnosis at the table was probable cancer of the pancreas obstructing both the duodenum and the common bile duct.

The fundus of the gall-bladder was anastomosed to the anterior wall of the stomach in the prepyloric region. A posterior contraperistaltic gastrojejunostomy was done. The patient recovered.

He returned to the hospital in 1932 to be treated for food poisoning. In 1934, he returned again because of rupture of the columnae carnae or chordae tendinae. Again in the same year he was treated for acute cardiac dilatation. In 1935 he sustained an injury in an automobile accident and was brought to the hospital with fracture of the ribs, lung injury, fracture of the radius and mild concussion. He returned again in 1936 with novocaine poisoning following tooth extraction, and again that same year, for auricular fibrillation. In March of 1937 he was treated at the hospital for diarrhea, and two months later for cardiac dilatation which caused death.

The post-mortem revealed a thick-walled gall-bladder, empty, anastomosis with good stoma about 5 mm. in diameter, the pancreas normal with no increased firmness.

This is just one example, but in my experience there have been many others in which it was utterly impossible to make a diagnosis without an operation, and although (as is shown by the history) the correct diagnosis was not made even at operation, yet a therapeutic measure was instituted then and there which, as far as I know, was the simplest, safest, and most logical that could have been carried out with any hope of making the patient more comfortable should he prove to have cancer of the pancreas, or of curing the condition should it prove to be chronic pancreatitis.

Again comes the patient who has had an epileptic or epileptoid seizure and is found to have hypoglycemia. There is no way of determining the presence or absence of an adenoma of the pancreas except by operation; and no way of curing the case without operation should an adenoma be present. An exploratory laparotomy is indicated.

Certain cases of ascites in women are due to rupture of an ovarian cyst. Papilliferous carcinoma or even pseudomucin escapes into the peritoneal cavity and ascites is the result. In either event the best treatment is early removal of the cyst, and often only by exploratory laparotomy can early diagnosis be made. Or, if it were ascites due to liver cirrhosis, it has been proved over and over that the Talma Morrison procedure, if used early, is most beneficial. A contraindication to the operation in ascites would be the ascites due to heart failure, or heart failure in any kind of ascites.

There is another set of circumstances in which I think early exploration should be the rule, and only the surgeon in the case can know how much one hates to have to perform such an operation. This is a postoperative exploration.

Let us suppose the patient has been making a normal recovery from, or convalescence after, his operation. Suddenly something happens which should not happen in normal conditions. Pain, collapse, nausea with or without vomiting may occur. Flatus may cease passing or the patient may visibly bleed. Any one of these symptoms or signs means that there is some serious deviation from the normal. The time to make the diagnosis and administer proper treatment is in the early hours. In the first twenty-four hours after operation, it is true, collapse may be a signal of failing heart in which case an operation would probably cause death. Consequently, no exploratory operation should be performed under these circumstances. When, however, acute cardiac failure can be excluded, when one can be certain that the cause of the symptoms is intra-abdominal, it is the part of wisdom not to hesitate, but to operate at once.

SUMMARY

There are very clear-cut indications for exploratory laparotomy. With all of the diagnostic aids in use at present and even with the maximum amount of skill on the part of the physician, conditions arise which are not possible of diagnosis without exploratory laparotomy.

Care is needed lest exploratory laparotomy be performed when the lesion is elsewhere than in the abdomen.

In the chronic cases, all of the evidence available otherwise must be obtained. Such a case is usually not urgent and there should never be haste in deciding to explore.

The acute surgical abdomen and all that is comprised under this title is of such a nature that exact diagnosis cannot be made in time to save the patient's life. Early exploration should be the rule.

Every instance of suddenly appearing stomach trouble in a patient over forty-five in which diagnosis cannot be made or which does not respond to proper medical treatment within a period of four weeks, should be subjected to exploratory laparotomy. To wait for the *x*-ray to make certain of the diagnosis will often lead to the death of the patient. In the present condition of our ignorance, exploration of all stomach cancer suspects will do for cancer of the stomach what has been done for breast cancer.

The jaundiced patient who has never had a history of gallstones and who has jaundice without pain may not have cancer of the pancreas, all opinion to the contrary notwithstanding, and his condition may be relievable by surgical means. An exploratory laparotomy is indicated.

Any penetrating wound of the abdomen should be explored immediately. Any other abdominal injury which fails to improve after six hours should be subjected to exploration.

No exploratory laparotomy should ever be done merely for the sake of making the diagnosis or of confirming a hopeless one. The reason for the performance of the operation must always be for the benefit of the patient.

SCLEROTHERAPY FOR PILONIDAL CYST*

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THE success of sclerosive therapy in varicose veins has stimulated the reapplication of this method of treatment to allied conditions. The injection treatment of bursae, hemorrhoids, hydrocele, and hernia, represents some of the newer trends in chemical surgery. In 1933 Cutler and Zollinger[1] revived the ancient method of treating pilonidal cyst by the use of irritating solutions. Their success prompted me to study this problem, and the results obtained justify the report on which this article is based.

The term pilonidal was applied to this developmental anomaly by Hodges. Actually, it means a nest of hair. These hairs frequently protrude from sinus openings, or may be found at the cystic dilatation which is frequently present. It is now recognized that these congenital depressions are due to faulty fusion of ectoderm in the sacrococcygeal region. It is not generally known that almost one-third of infants, at birth, show a sinus in this area. Usually it disappears with growth, but in about 4 per cent of cases, especially in males, the defect persists.

The sinuses may be single or multiple, and vary in depth from a few millimeters to more than an inch. Cystic dilatations may be present at any point of the sinus; usually at the end of the passage. When multiple sinuses are present, they will be found to communicate with the same blind cystic pouch. It is important to keep this fact in mind during the treatment.

It is interesting to note the ancient origin of sclerosive therapy, and its recent revival. As far back as 1829, Dzondi[2] treated a fistula of the neck by injecting liquor nitrici hydrargyri. Apparently this method of treatment proved discouraging, because there is no evidence of its continuation in the literature of that time.

The neurologic surgeons must be credited with the stimulus for the modern approach to the problem. It had been noticed that solutions of the type of Zenker's[3] and Carnoy's[4] would fix the lining of certain forms of gliomatous cysts. As a result, the tissues became tougher, and were more easily removed surgically. In addition these solutions showed definite hemostatic qualities. These properties were seized upon by Cutler and Zollinger for their treatment of cysts and fistulae.

The possibility of replacing surgery by a simple sclerosing procedure led me to attempt the duplication of Cutler's method. In the procedure as outlined by the authors, the abscesses were first incised and drained. Following this, modified Carnoy's solution was applied to the abscessed cavity, which was subsequently curetted. The sinus tracts were also laid open, after which the cauterizing solution was applied to the lining membrane. This was again followed by curettage and further chemical treatment. Their method gave good results. However, it involved open surgery, which, although of a minor

[1] CUTLER, E. C., and ZOLLINGER, R. The use of sclerosing solution in the treatment of cysts and fistulae. *Am. J. Surg.*, 19: 411 (March) 1933.

[2] DZONDI, C. H. On congenital fistula of the trachea. Schwetsche & Son, 1829; Reproduced in the British Record of Obstetric Medicine & Surgery for 1848, vol. 1.

[3] Zenker's Solution: A fixative solution of corrosive sublimate (5 parts); potassium bichromate (2½ parts); sodium sulfate (1 part); water (100).

[4] Modified Carnoy's Solution: absolute alcohol—6 c.c.; chloroform—3 c.c.; glacial acetic acid—1 c.c.; ferric chloride—1 Gm.

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nature, was the very thing I wished to avoid.

Of the three cases reported here the first had a frank suppurative process and was incised. The other two patients had no abscess and were successfully treated without incision.

Fig. 1. Infected pilonidal cyst incised, cavity packed with iodoform gauze. Note gauze in abscessed cavity.

TECHNIQUE

Patients will usually report for treatment after infection has occurred. However, in some cases, the presence of slight swelling, or history of a discharge, may bring the patient under observation. Routine physical examination may also disclose the presence of small sinuses before symptoms appear. This technique may be applied at any of these stages.

The rare condition of spina bifida should be ruled out before attempting treatment. The remote possibility of a congenital cyst communicating with the dura or a meningocele can be excluded by simple *x*-ray examination. The area should then be closely studied to learn the location of all the sinus openings. Sometimes, during treatment a tract will be disclosed which was invisible at the outset. Infected areas should be incised and drained before sclerosive therapy is instituted.

The sinuses must first be probed until they are sufficiently wide to admit very fine curettes. Instead of probes, fine needles may be employed which have had their tips dulled by grinding. Probing should be continued until the common cystic enlargement of the sinuses is found. Modified Carnoy's solution is then injected, using a dull needle which is inserted into one of the sinuses. Enough solution is injected to fill all the tracts. At the next treatment each tract is curetted with a very fine curette or the following substitute: A needle with a long bevel is deliberately bent so that the tip becomes deflected to form a slight burr or hook. This catch on the needle tip may be employed as a curette by being run forwards and backwards in the sinus canal. It is advisable to use preliminary anesthesia. Two per cent nupercaine solution should be carried into each sinus on a fine cotton-tipped probe.

Following the curettage, the sclerosing agent, fuming nitric acid is applied. At the outset of this technique I employed the modified Carnoy's solution advocated by Cutler and Zollinger. However, it was soon discovered that this preparation did not have enough sclerotic power for this

procedure in sinuses. Apparently a stronger caustic was necessary, and fuming nitric acid was decided upon. It is applied on a probe or fine metal applicator, which has been dipped into the acid, and the excess drops removed. A thin film of acid remains on the metal tip of the instrument. After curettage this is inserted to the depth of the canal, and the metal rotated, bringing the solution in contact with the entire circumference of the lining cavity. Curiously enough this procedure is not painful, the patient experiencing only a slight burning sensation. Treatments are given every two to four weeks until complete obliteration ensues.

FIG. 2. Healed. Note scar tissue at site of abscess and sinus openings.

CASE REPORTS

CASE I. Age 16. An abscess had been present in the sacrococcygeal region for one week. The swelling was incised under local anesthesia and the cavity packed with iodoform gauze. (Fig. 1.) Three days later the abscessed cavity was smaller, and was cleaned. Modified Carnoy's solution was applied for ten seconds on a cotton-tipped applicator. Two sinuses were discovered leading into cavity. Two weeks later the cavity was clean and almost closed. Three weeks later three sinus openings were located, curetted, and treated with fuming nitric acid. The largest sinus, when probed, was found to be 2 inches long. Long hairs were removed when this sinus was curetted. Two weeks later curettage and cauterization were performed. One month later one sinus closed and the remaining two were treated. One month later the largest sinus was still slightly open. It was curetted and nitric acid applied. One month later the pilonidal cyst was completely cured. The sinus openings exhibit firm scar tissue; the probe cannot be pushed in. (Fig. 2.) A check-up one year later disclosed no recurrence.

CASE II. Age 30. Patient complained of a discharge on underwear, slight itching, and discomfort in the sacrococcygeal region. Examination disclosed a pilonidal cyst with two sinuses. No active infection was present. It was decided to institute sclerotic treatment immediately after the sinuses were probed. After two preliminary probings, to ascertain length and direction of sinuses, the first treatment was instituted. Modified Carnoy's solution was injected through the sinus opening, using a dull-tipped needle. Two weeks later both sinuses were curetted (under nupercaine anesthesia)

with the removal of numerous hairs and lining membrane. Following this, fuming nitric acid was applied on an ordinary metal applicator tip, deeply into both sinuses. The sinus openings were dressed with a small gauze pad containing boric acid ointment.

Two weeks later the same procedure was performed. One month later, on attempting curettage, it was found difficult to enter the sinus tracts, because of beginning obliteration. The probe containing the nitric acid was admitted partly into each tract. A dry dressing was applied. One month later both sinus openings were completely closed. A check-up one year later disclosed no recurrence.

Case III. Age 33. This patient complained of itching and discharge in the sacrococcygeal region for several years. Examination disclosed a pilonidal cyst with two sinus openings. These were probed to the point of common intersection several times, then modified Carnoy's solution was injected. One week later the sinus tracts were curetted, using a needle which had been deliberately bent at its tip. Fuming nitric acid was applied on a probe into each sinus tract. Two weeks later another treatment was given. The third treatment was given one month later. The sinus openings were smaller and difficulty was experienced in introducing the probes. When the patient was seen one month later, both sinus openings were firmly closed, and the patient discharged. Six months later the patient was re-examined with the same findings.

SUMMARY

1. A modification of the method of Cutler and Zollinger for the injection treatment of pilonidal cyst is presented.

2. The method is non-surgical and depends upon the cauterizing action of fuming nitric acid when it is applied to the sinus tracts.

3. Check-up examination after a year discloses the permanency of the end result with this method.

THE USE OF ALKYL-DIMETHYL-BENZYL-AMMONIUM CHLORIDE IN INJURY*

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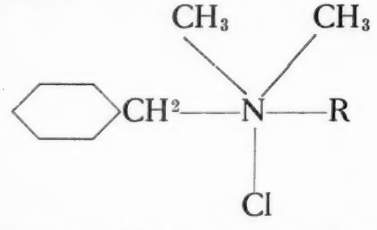
IN 1935, Gerhardt Domagk[1] introduced an entirely new type of germicide. The compound is a mixture of alkyl-dimethyl-benzyl-ammonium chlorides in which the alkyl group represents a series of homologous radicals derived from the fatty acids of coconut oil. It is a brand of dial konium (zephiran). While the antiseptic properties of coconut oil are well known, the combination with quarternary ammonium base had theretofore not been investigated.



The compound is an amber-colored solid of soap-like consistency, which is quite uniform in composition. It is soluble in water, alcohol and acetone. Solutions in germicidal concentrations impart a soap-like sensation to the skin, leaving it smooth and satiny. Germicidal solutions for clinical use have a surface tension of less than 36 dynes per c.c., as compared to distilled water which has a surface tension of 70 dynes per c.c. at room temperature.

Extensive pharmacologic and toxicologic studies were carried out by Deskowitz.[2] Solutions of 1:1000 were given by mouth to guinea-pigs as their only source of fluid for months without any deleterious effects upon health. Microscopic examination of the organs was negative. A 1:1,000 solution injected intraperitoneally (up to 6 c.c.) daily for several months caused no reaction, while more concentrated solutions caused a fibrinous reaction. A 1:1,000 solution applied to a rabbit's shaved ear and allowed to dry, revealed no irritation. The 1:1,000 dilution in the form of gauge packs was the highest concentration that could be applied for twenty-four hours to the ear with slight evidence of irritation resulting.

Dunn[3,4,5] and Heineman[6] reported that alkyl-dimethyl-benzyl ammonium chloride possessed a high germicidal potency against a variety of pathogenic organisms. Penetration and inhibition tests, conducted by Heineman, were made according to the agar cup-plate method of the United States Government Food and Drug Administration. Staphylococcus aureus was inhibited in a concentration of 1:100,000 both in plain and serum agar. E. typhosa was inhibited in a concentration of 1:5,000 but not in a concentration of 1:10,000.

White[7] and his co-workers found in clinical study that there was a marked reduction of infection incidence in laparotomy cases when this antiseptic was used, as contrasted with the cases in which 3½ per cent iodine was used.

The present study was undertaken by the Traumatic Service of Harlem Hospital during the months of October, November and December, 1937, and January, 1938, with regard to the efficacy of alkyl-dimethyl-benzyl ammonium chloride in the care and treatment of injuries. Over 300 cases were treated, but 177 completed cases are presented as a preliminary report. These include:

A. Skin preparation for the following:
   1. Exploratory laparotomies for penetrating stab wounds and bullet wounds of the abdomen.

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2. Exploratory operations following stab wounds of the neck.
3. Removal of foreign body from the neck.
4. Removal of foreign bodies from the extremities.
5. Open reduction of fractures of the extremities, with application of Lane plates and insertion of bone graft.
6. Lacerations of the extremities with repair of nerves, tendons, and vessels.
7. Repair of lacerations, both superficial and deep, of scalp, face, neck, and extremities.
8. Amputations of extremities.

B. Treatment of:

1. Burns.
2. Infected lacerations, deep infections, and cellulitis.
3. Avulsions.

A definite routine was adopted with regard to the technique of skin preparation, the type of solution to be used, and the dilutions necessary.

WOUNDS

For the disinfection of wounds or traumatic injuries, or as a first aid prophylactic, the tincture, 1:1000, was used.

For deep lacerations where the application of alcohol should be avoided, the aqueous solution, 1:5,000, was used.

For widely denuded areas, for wet dressings, and for irrigations of deep infections and sinuses, the aqueous solution, 1:5,000, was used.

PREOPERATIVE SKIN PREPARATION AND DISINFECTION

Ward Preparation. When a patient was prepared on the ward for laparotomy, etc., the field was shaved, washed with water, alcohol, and ether, then with the antiseptic in aqueous solution, 1:1,000, which was allowed to dry.

Operating Room Preparation. On the operating table the field was again washed with alcohol and aqueous solution of zephiran, 1:1,000, alternately two or three times in six to eight minutes. When the patient was draped, the area was painted with the tinted tincture, 1:1,000.

PREPARATION FOR OPEN BONE OPERATIONS AND JOINT OPERATIONS

The same preoperative skin preparation was carried out (i.e., with water, alcohol, ether, then with alkyl-dimethyl-benzyl ammonium chloride, aqueous solution, 1:1,000). This preparation was done daily for three days, and then the operating room routine was carried out as stated above.

REPAIR OF LACERATIONS

Eighty cases of deep and superficial lacerations of the scalp, face, neck, and extremities were selected for study. Some of these were on the ward because of associated injuries. They varied from superficial scalp and face wounds to deep lacerations of the face, neck, and extremities, of which some were very extensive. The technique was to apply zephiran tincture, 1:1,000, into the wound and to the skin surfaces in the more superficial injuries. In the deep lacerations, we employed a primary irrigation with the aqueous solution, 1:1,000 or 1:5,000, depending upon the extent of the lesion, and painted the skin edges with the tinted tincture, 1:1,000. Some of these wounds were sutured, others approximated with adhesive strips. In only one case, a deep laceration of the forearm, did infection result. In this instance, following removal of the sutures and free irrigation with the aqueous solution, 1:5,000, the infection promptly cleared. The fact that zephiran is not irritating, when used in proper concentrations, and does not damage the healing surfaces makes it particularly efficacious in this type of injury.

BURNS

The Harlem Hospital technique in the treatment of burns is a primary debridement, followed by the application of an eschar of tannic acid, followed by 10 per

cent silver nitrate. When this eschar begins to separate, it is gradually debrided. After debridement, the denuded surfaces often become infected from unavoidable contamination. In fifteen cases zephiran aqueous solution, 1:5,000, was applied in wet dressings following the above debridement. There resulted a rapid recession of temperatures from 101 to 103 degrees to normal. Healing then rapidly resulted, first with healthy granulations, then good epithelium. In some of the cases the total skin involved in first, second, and third degree burns varied up to 40 per cent. A solution of 1:10,000 is recommended in infants and children. It is our opinion that the results in burns are striking.

AMPUTATIONS

In four cases, middle thigh amputations were done because of gangrene, either of diabetic or arteriosclerotic origin. In all four there was good union at the stump with no subsequent infection. Noteworthy is the fact that these cases fell in the advanced age group.

THE ABDOMEN

Through the four months period in which this study was conducted, there were thirty-six patients who required exploratory laparotomy because of penetrating stab and bullet wounds of the abdomen, excluding those in whom death occurred from shock and hemorrhage too immediate to determine any evidence of abdominal wall or intra-abdominal infection. Eighteen of these were prepared by the above mentioned zephiran technique. All were emergency operations. The alternate eighteen received the usual iodine-alcohol technique. No abdominal wall infection resulted in any of the cases, and no intra-abdominal infection could be traced to faulty skin preparation. In both groups, only the patients with bowel perforations contracted peritonitis, a complication which was usually anticipated, but which varied in severity. In six additional cases exploration was done down to the peritoneum but no penetration was observed. Three of these explorations were carried out under zephiran technique. No abdominal wall infection resulted. In many instances profound hemorrhage, as a result of laceration of the spleen, liver, or the mesentery vessels, often left the patients in a weakened postoperative state, but no abdominal wall infection resulted even in these cases.

OPEN BONE OPERATIONS

There were five open bone operations without previous infection or potential infection; this would eliminate the cases of acute osteomyelitis. A large number of cases of compound fractures, especially those of the long bones of the extremities, cannot be considered because studies of a different character are now being carried out by the Traumatic Service on this particular group. Three open reductions of fractures of long bones were done with the zephiran technique.

Case 1. In an adult with fracture of the shafts of the radius and ulna, Lane plates were applied; primary union resulted with no evidence of bone infection.

Cases II and III. Two children had open reductions of fractures of the tibia and fibula, with the application of Lane plates to the tibia; both had mild superficial infection. In all fairness, other factors entered. One had poor skin but operation was deemed most necessary. In both instances the operation was performed by teams not by long experience versed in exacting bone technique. This is apt to be the case in a large general hospital with frequent changes in service.

Another patient had a bone graft inserted into the tibia, with primary union and no evidence of bone infection. The last patient had an open operation of the lower end of the humerus, with the removal of badly displaced comminuted fragments. Primary union resulted.

While the authors feel very definitely that the zephiran technique may with every safety be used in open bone operations, a larger series is recommended before this can be proved conclusively.

THE NECK

Ten cases were explored for possible laceration of the greater vessels. One had a complete severance of the internal jugular vein. The skin was prepared with 1:1,000 tinted tincture. The exploration of necessity invaded the deep fascial planes, but no infection resulted.

Case I. A deep, self-inflicted 3 inch laceration of the upper anterior surface of the neck, down to and involving the posterior wall of the pharynx. No damage had been done to the great vessels. A tracheotomy was done and the wound repaired with the zephiran technique. No infection resulted.

Case II. A bullet entered the mouth and lodged in the middle of the neck on the right side in the deep structures. The patient was operated upon one week after injury for removal of the bullet. It was found adjacent to the carotid sheath on the posterior and mesial surfaces. Skin preparation consisted of zephiran technique on the ward and the use of zephiran tinted tincture 1:1,000 at the time of operation. No infection resulted.

FOREIGN BODIES IN EXTREMITIES

Alkyl-dimethyl-benzyl ammonium chloride was used for skin preparation for the removal of foreign bodies in five cases. All of them were needles, three imbedded in the hand and two in the thigh, one in a 3 months old infant. None of the patients evidenced any postoperative infection and all healed with primary union.

REPAIRS OF LACERATIONS OF TENDONS, NERVES, AND VESSELS

There were eighteen cases of tendons of the forearm and hand repaired after zephiran preparation. Fifteen of these were discharged to the Out-Patient Clinic at periods varying from four to seven days following admission, with no evidence of infection. Three had mild infection. These three were found to have delayed entry into the hospital, with increasing chance of infection and tendon necrosis. In all the cases the aqueous solution, 1:1,000, was poured into the open wound for its peroperative germicidal action. The results of this series are to be considered excellent. Three of the above cases had nerve involvement, and repair was accomplished at the same time. In all the cases, vessels were severed, varying in importance, up to the radial artery, and simple ligation was resorted to. There was also a series of eight cases with laceration of the major vessels without tendon involvement, two of these involving the brachial artery. The results of these cases were equally good. There was one case in which the musculospiral nerve was freed from scar tissue. Primary union with no infection resulted.

INFECTED LACERATIONS, DEEP INFECTIONS, AND CELLULITIS

This group was rather limited, except in cases admitted with neglected lacerations, hand infections, human bites of the fingers, and a few cases of cellulitis of the extremities.

Human bites are a treacherous group of cases and offer no fair test for the germicidal properties of any drug. Infected lacerations responded very nicely to either wet dressings of the aqueous solution, 1:5,000, or with irrigations with this dilution. Its initial use in any infection depends upon the possibilities of irrigation. It was found to be of much benefit in clearing up the infection following free incisions. Deep irrigation with zephiran aqueous solution, 1:5,000, or massive wet dressings with the same dilution, are recommended. Its use in cellulitis comes mostly after incisions for drainage following localization. It is to be noted that no brief is made for this germicide in either hand infection or cellulitis, as an aid towards localization or infection. Hypertonic qualities are not claimed. Its work starts following incisions for drainage. The skin was prepared for incision with the tinted tincture, 1:1,000.

AVULSIONS

The aim in these cases must be towards the prevention of infection. This calls for

necessary debridement under adequate anesthesia, and the use of drugs of high germicidal potency, to be followed by wet dressings with an antiseptic which will not irritate the denuded surfaces. In four cases, following debridement, tinted tincture of zephiran, 1:1,000, was used and followed by continuous wet dressings of the aqueous solution, 1:5,000, until the danger of infection had passed. The results were very pleasing. Five other cases in which the initial application was not tincture of zephiran also healed nicely under continuous wet dressings of the aqueous solution, 1:5,000.

CONCLUSIONS

1. The authors are convinced that alkyl-dimethyl-benzyl ammonium chloride has a very definite place in the germicidal and antiseptic armamentarium for combating infection in injuries. In proper dilutions it does not damage tissue. It thereby enhances the possibility of rapid healing.

2. Its potency is manifested even in the weaker dilutions, and it can be used on large denuded surfaces with comfort to the patient and without toxic effects.

3. Its use in preparation of the skin for operation involving the abdomen and neck, and its use in bone surgery with a minimum of infection, gives some evidence of its high germicidal potency.

4. Its use in burns has given good results in combating secondary infection.

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CASE REPORTS

PERSISTENT HEADACHE RESULTING FROM DISPLACEMENT OF THE HEART AND MEDIASTINUM RELIEVED BY PHRENIC INTERRUPTION

REPORT OF A CASE

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THE following case is of interest because of the unusual findings and the apparent value of phrenic interruption in relieving the symptoms.

The patient was a young woman aged 25 years. She complained of severe headaches of about eight years' duration which were becoming progressively worse. The pain would start in the right side of the chest and shoot up the right side of the neck and head. The patient stated that she obtained relief from her pain when lying down but that it returned immediately upon arising.

She had had an operation for appendicitis shortly before the headaches started. There was nothing significant about the operation except that she remembered being in bed a long time and feels that she did not get along well. The headaches were not related to her menstrual periods. The only other significant point in her history was that she had some dyspnea and tachycardia on exertion.

Examination of the patient revealed a well developed and fairly well nourished young woman 5 feet 6 inches tall weighing 120 pounds. Her blood pressure was 110 systolic and 70 diastolic; pulse rate was 86 per minute and temperature was 98.2 F. Her uterus was retroverted but otherwise normal. Examination of the nose and throat showed slight deviation of the septum but no evidence of pathology to explain the headaches.

Roentgenograms of the chest showed the heart and mediastinum to be pulled over to the left. From the fifth rib to the base the lung field demonstrated a homogeneous haziness. The condition was thought to be atelectasis of the left lower lobe. (Fig. 1.)

Because of the marked displacement of the heart and mediastinum a temporary phrenic interruption was done on the left side. With the rise of the left hemidiaphragm the heart and mediastinum returned to a more normal position. (Fig. 2.)

Following the left temporary phrenic interruption the headaches disappeared at once and the dyspnea, which was more pronounced immediately after the phrenic, disappeared in about a month. The patient was examined at monthly intervals, and there was no return of the headaches for six months. She has been perfectly well with the exception of some decrease in appetite as a result of the phrenic interruption.

Between the sixth and the eighth months headaches recurred on three occasions. Fluoroscopic examinations at the end of the seventh and eighth months after the operation showed that the diaphragm was no longer elevated and the function was returning.

Eleven months after the phrenic operation Dr. Schneiders of San Diego was consulted and he reported that the headaches were becoming more frequent and more severe. A bronchoscopic examination showed the left main bronchus to be pulled to the left so that it was difficult to see around it. However, an aspiration probe was passed beyond this point about 3 inches. The deviation was thought to be due to airlessness or atelectasis of the lower lobe.

COMMENT

A case of severe and persistent headache resulting from displacement of the heart and mediastinum and relieved by phrenic

interruption is reported. It was felt that the lesion was an old atelectasis, probably on the basis of a fibrous stenosis following a pulmonary infection after the appendectomy eight years previous. That the headaches were associated with the intrathoracic lesion was demonstrated by the fact that they disappeared when the left hemidiaphragm was paralyzed and elevated, and that they returned when the function of the diaphragm returned. The elevation of the left hemidiaphragm resulted in some return of the mediastinum to a more normal position, thus releasing the tension upon the vessels or the sympathetic or vagus nerves.

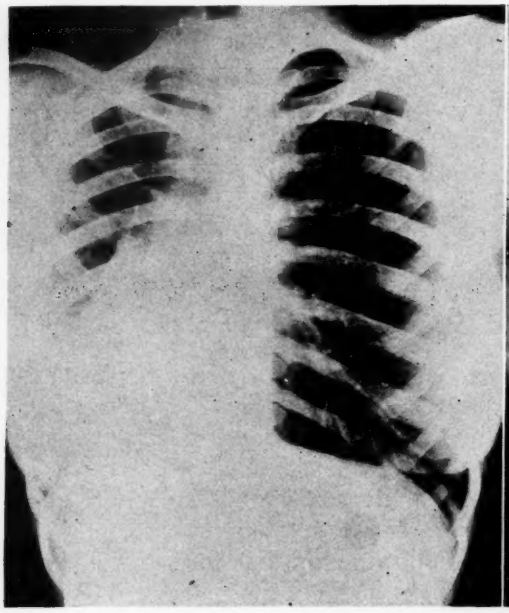

FIG. 1. Roentgenogram of chest before phrenic interruption. Note the displacement of the heart and mediastinum to the left. Also note the displacement of the trachea.

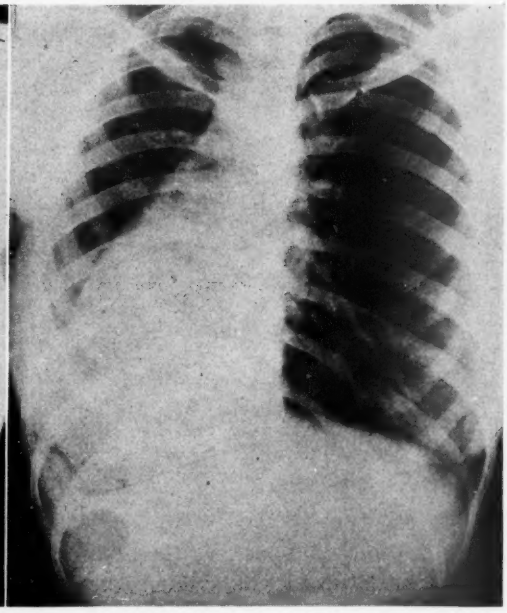

FIG. 2. Roentgenogram of the chest after phrenic interruption. Note the elevation of the left hemidiaphragm. Also note the more normal position of the heart and mediastinum.

Banyai reported a case in which severe facial neuralgia was relieved by phrenic nerve block. However, the pathology in his case was entirely different in that his patient had advanced pulmonary tuberculosis with diaphragmatic pleurisy.

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POSTOPERATIVE ABDOMINAL EVISCERATION

REPORT OF SIX CASES

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THE problem of disruption of postoperative wounds confronts every surgeon who performs a large number of laparatomies. Until recently few writers had reported many of these cases, but of late there have been a considerable number described. As the condition is always serious and carries a high mortality, its early recognition is essential.

A recent experience with a concealed type of evisceration presented a problem the clinical features of which were important enough to warrant careful study, and together with five other personal cases, to prompt the preparation of this report. The details of one case are given in full; the others will be briefly mentioned.

CASE 1. H. B., male, aged 57, was admitted to Madison Park Hospital, August 4, 1937, with a complaint of pain in the right lower part of the abdomen, accompanied by vomiting. The preoperative diagnosis was acute suppurative appendicitis.

Operation. August 4, under spinal (novocaine) anesthesia, supplemented by gas-oxygen and ether, a right rectus incision was made. The rectus was not split, but retracted laterally. The appendix, 3 inches long, was found in the retrocecal position. The tip was gangrenous and attached retrocecally to the posterior surface of the liver. Free fluid but no pus was found within the peritoneal cavity. The appendix was removed in a retrograde manner, the stump cauterized with pure phenol, but not inverted. Closure was performed in separate layers, using plain gut for the peritoneum, chromicized gut for the fascia and silk for the skin. No retention sutures were used. During the closure a small area of the small intestine was accidently pinched by a clamp. This area was inverted and buried by means of a purse-string suture.

Postoperative Course. From August 4 to August 9, the course was uneventful. Temperature, pulse and respiration were normal. On the latter date at 5 P.M. vomiting began and the temperature rose to 102.4°F. Pulse was 120, respiration 20. The patient showed slight distention and complained of epigastric pain. An intravenous injection of 5 per cent glucose was administered, and a complete blood count and chest *x*-ray were ordered. A blood chemistry was also done. The abdomen was soft, there was no cyanosis of lips or fingertips, and the wound was clean. An enema was effectual.

On August 11, temperature, pulse and respiration were better; white blood count and differential counts were normal; urea nitrogen was 30. The chest *x*-ray proved negative. The urine was normal and the bowels normal with enemata. The next day the patient complained of slight nausea, but otherwise felt much better.

As a matter of differential diagnosis, the four following conditions were considered (among others) and ruled out: (1) heat stroke; (2) minute pulmonary embolism; (3) partial collapse of the lung; (4) pyelitis (because of burning upon urination).

The wound was clean. A few sutures were removed, and the interior of the superficial part of the wound was examined. There was very poor healing and the edges separated easily. Slight edema was noted. The wound was strapped.

Following another period of distention during the previous night, the wound suddenly burst open on August 14. The intestines were found extruded upon the skin over the abdomen. At 5.15 A.M., the patient was in a condition of partial shock. Secondary operation was performed under gas-oxygen anesthesia. The exposed intestines were bathed with normal saline and replaced. Inspection of wound and peritoneal cavity revealed no pathologic condition except a slough of the fascia and poor healing. The wound was closed by means of through-

and-through sutures of heavy silk. The condition of the patient was poor and evidences of shock were still present. A blood transfusion was given and supportive measures were instituted, but in spite of everything the patient failed to react and died at 8 P.M.

Query. Was there a partial or concealed evisceration on August 9 and thereafter?

In retrospect, one is inevitably led to such a conclusion after seeing the disrupted wound. The edges and side of the wound showed practically no evidence of healing. The fascia had not united at all, the edges having sloughed away. In spite of the fact that the intestines did not appear to be strangulated or obstructed, no marks of constriction having been found, the mild syndrome on August 9 was unquestionably that of intestinal obstruction. The institution of Wangensteen suction with marked improvement in all symptoms and the lack of swelling over the wound led us into the belief that an early intestinal obstruction due to adhesions from the appendix stump or elsewhere had been averted.

On August 12, however, eight days postoperative, poor healing was noted after removal of two sutures in the upper part of the wound. Because of this observation the remaining sutures were left in and the wound strapped. On this occasion, had the remaining sutures been boldly removed and a more thorough inspection of the wound made the sloughing fascia and perhaps a tongue of omentum or a loop of gut might have been seen through the parting fascia. At this stage the patient was in excellent condition and such a discovery followed by prompt remedial efforts might have resulted in an entirely different outcome.

Case II. A child of 5 was operated on for acute suppurative appendicitis with perforation and abscess. On the fifth day, following a coughing spell, several loops of gut appeared under the dressing. The child was in partial shock. Under aseptic precautions, the gut was replaced, the wound packed with iodoform gauze and strapped tightly. Recovery followed.

Case III. A female aged 35 was operated on for fibroids (hysterectomy). Her general condition was apparently good, but on the eighth day following the removal of sutures, she became nauseated and vomited shortly after a bowel movement. Examination of the wound showed complete dehiscence with a large tongue of omentum protruding from a partly opened skin incision. This was returned to the abdomen and the wound packed with a wide thick strip of iodoform gauze and closed except at one end with adhesive. Recovery took place.

Case IV. A ruptured gastric ulcer in a man of 61 was repaired under spinal anesthesia by plication using Pagenstecher linen. Omentum was used to cover the repair of the ulcer. The wound was closed in layers using one Penrose drain. Retention sutures of silkworm gut were used. This patient had marked arteriosclerosis. He was restless and at times seemed delirious. Because of his attempts to get out of bed, sideboards were applied. However, during the night of the twelfth postoperative day, the patient climbed out of bed and collapsed.

Examination showed a ruptured abdominal wound with bowels protruding and lying all over the abdomen. One loop of small intestine (6 inches long) had been almost completely torn off its mesenteric attachment and was adherent in the abdomen to the right side of the incision. It was raggedly torn wide open. The abdomen and intestines were badly soiled. There was very little bleeding. Patient was in shock. Immediate supportive measures were instituted, including blood transfusion. Without anesthesia, a resection and side-to-side intestinal anastomosis after proper cleansing of the operative field were performed. The wound was closed with interrupted heavy through-and-through silk sutures, using two Penrose drains. The patient died thirty-six hours later.

Case V. A ruptured duodenal ulcer in a white man of 32 was given the usual type of repair under spinal anesthesia. Silkworm gut retention sutures were used. Convalescence was uneventful until the fifth day when after a coughing spell patient felt nauseated and thought he "busted" his wound. Examination showed partial rupture of the wound with a loop of gut presenting, adherent to one side. The wound was packed with iodoform gauze and tightly strapped, and the patient recovered.

Case VI. An incisional hernia in a man of 28 had been operated on twice before. Under spinal anesthesia the peritoneum was separately closed with plain catgut followed by a vertical overlap repair using No. 3 chromic gut, with silk for the skin. During the night of the seventh day following removal of the skin sutures, the patient coughed, felt sharp pain in the abdomen and vomited. Examination

showed complete evisceration with several loops of small intestine out on the abdomen. The general condition of the patient was excellent.

Under gas-oxygen ether the abdomen was properly prepared, the intestines replaced and the abdomen closed with through-and-through interrupted double heavy silk sutures. A thick layer of iodoform gauze was placed upon the abdominal contents just under the closure and allowed to come out at one end. This was removed in stages after the seventh day. The sutures were removed, a few at a time after the twelfth day. Recovery was uneventful.

SUMMARY

No.	Sex	Age	Operation	Anesthesia	Day of Rupture	Nature	Type of Repair	Remarks
1	Male	54	Appendectomy. No drainage	Spinal with gas and oxygen	14	Complete	Through-and-through silk	Shock, death. Probably incomplete for 4 days. Long operation G. & O. had to be supplemented. Closure difficult
2	Male	5	Appendectomy	Ether	5	Complete	Iodoform gauze packing; strapping	Recovery. Closure done in patient's bed.
3	Female	35	Hysterectomy	Gas-oxygen ether	8	Incomplete	Iodoform gauze packing; strapping	Recovery
4	Male	61	Laparotomy for ruptured gastric ulcer. Drainage	Spinal	12	Complete	Through-and-through silk; intestinal resection; anastomosis	Death
5	Male	32	Laparotomy for ruptured duodenal ulcer. Drainage	Spinal	5	Partial	Gauze and strapping	Recovery
6	Male	28	Incisional hernia	Spinal	7	Complete	Gauze and through-and-through silk	Recovery. Sutures out on fourteenth day.

COMMENTS

While this series is too small to form any definite conclusion, there are, nevertheless, several points of interest:

1. Mortality, two out of six cases, or 33⅓ per cent.

2. Only the elderly patients died.

3. Clean wounds ruptured in the young as well as the old.

4. Incomplete evisceration may be very well managed at the bedside if done under suitable precaution.

5. McCauliff[15] found no case of rupture where a muscle-retracting incision was used. Case 1 in this series had this type of incision.

6. Alert, careful investigation of sudden changes in progress after operation may result in early recognition of impending, partial or concealed rupture. In such early recognition lies the hope and reward of a happy outcome.

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PULMONARY TUBERCULOSIS IN A CHILD

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THE case of a young Portuguese boy is presented here, not only because of its unusual character, but because in a brief span of life he has passed through the entire cycle of a dreaded disease. He has emerged for the present with the disease arrested, aided by modern therapeutic procedures. The prognosis is still problematical and time alone will tell the ultimate result. Because he has run the complete course from the pathogenic, diagnostic, and therapeutic viewpoints, he has offered at various times cross-sections of practically all stages and types. The illustrations offer the best medium for conveying this chronologic presentation.

CASE REPORT

On May 10, 1933, a 6 year old child, who had been born in Portugal, was referred to the lung clinic of the Babies' Hospital. There was a strong familial history of tuberculosis and the contact was both intimate and prolonged.

The child complained chiefly of cough, mucopurulent expectoration, and afternoon fever of several weeks' duration. Two months before the above syndrone, easy fatigability and malaise were noted by the grandmother. Anorexia and night sweats were more recent disturbances.

Physical examination revealed a slightly undernourished youngster, appearing somewhat chronically ill, and weighing 39 pounds. Beyond slightly infected tonsils, and poor teeth, positive findings were confined to the lung. Over the upper and mid-portions of the right lower lobe there were dulness, bronchovesicular breath sounds and constant medium inspiratory râles, persistent after cough. D'Espine's sign was negative.

An *x*-ray taken in April 1933, two weeks before admission, showed bilateral lesion of hilar adenopathy. (Fig. 1.) The process was somewhat more marked on the left side, and the diagnosis of primary infection was made. The Mantoux test—old tuberculin, dilution 1–1,000—was positive and gave a three plus reading.

Another *x*-ray, taken at the clinic of the Babies' Hospital, May 10, 1933, showed a remarkable change in the elapsed month. (Fig. 2.) The left hilar region seemed to be clearing and on the right side in the mid-zone extending from the hilum there was an extensive parenchymal process with a cavity whose diameter measured 2.5 cm.

A diagnosis of both primary type of infection and so-called adult tuberculosis (cavity formation) was made. The transition was so rapid that both phases could be noted at the same time. Hospitalization was advised with the thought of instituting collapse therapy.

On May 24, the patient was admitted to the hospital, and additional laboratory data instituted. His urine and blood counts were within normal range. Gastric contents, stools and a laryngeal smear all showed an abundance of tubercle bacilli.

Artificial pneumothorax was begun May 26, 1933. The average refill was 200 c.c., and treatment was repeated two or three times weekly. Collapse took place and the cavity became well defined. (Fig. 3.) About two months after the first artificial pneumothorax, the lung was collapsed to about 75 per cent and the cavity could be visualized as much smaller. (Fig. 4.) The mediastinum was pushed toward the contralateral side and a so-called mediastinal hernia could be detected.

It was decided to proceed with extreme caution. The question arose as to whether to insert some foreign substance, such as sterile mineral oil, into the pleural cavity in order to lend greater stability to the mediastinum. The boy was always difficult to handle, but at this stage he became more unruly than ever. Because he was so ungovernable, isolation was difficult and imperfect, and contamination of other small

children in the ward was feared. On July 31, 1933 he was therefore discharged to Glen Gardner Sanitarium, where better supervision and isolation could be maintained. Refills were given twice a week with great difficulty because of the child's temperament. Sedatives were of no avail and general anesthesia was the only possible way to maintain the pneumothorax. However, this was decided against, and artificial pneumothorax was abandoned because of the lack of coöperation. The patient continued to run a low grade temperature and to expectorate tubercle bacilli.

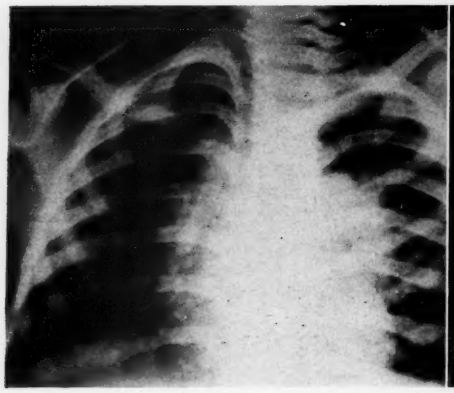

FIG. 1. April 1933. Bilateral hilar adenopathy, especially left.

FIG. 2. May 10, 1933. Left hilar adenopathy clearing. Extension into parenchyma of lung at right mid-zone with cavitation.

On October 17, a right phrenic nerve resection was performed. The diaphragm rose to nearly one interspace, but this had only a very slight beneficial effect upon the pathology.

The boy remained at the institution for about one year, during which there was a gain of 4 pounds. He did not run any temperature, but his sputum remained positive.

On July 18, 1934, he was admitted to the Essex County Isolation Hospital in Belleville, New Jersey. His sputum was positive for tubercle bacilli, his urine was negative, and blood Wassermann and Kahn tests were also negative. The blood count revealed 14,000 white cells, with 70 per cent polymorphonuclear cells, 29 per cent lymphocytes, and 1 per cent eosinophiles. Figure 5 represents the *x*-ray taken at that time. There was a slight rise of the right diaphragm as compared with previous films. At the site of the original lesion there was still infiltration and rarefaction suggesting the cavity. In the left upper lobe a recent exudate spread could be noted. The sedimentation rate averaged 80 (Westergren method), and the temperature fluctuated between 99 and 100.5 degrees.

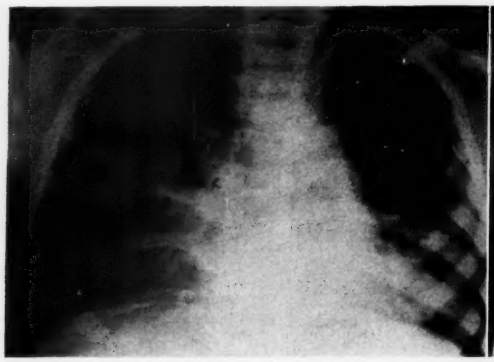

FIG. 3. May 15, 1933. Artificial pneumothorax instituted on right side. Cavity now well defined.

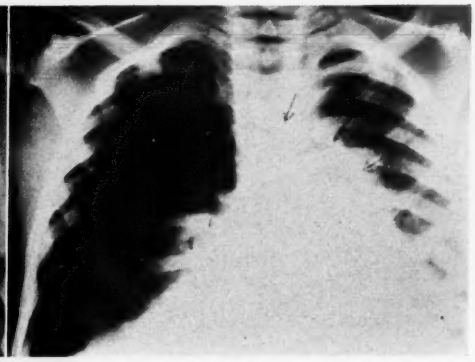

FIG. 4. July 1933. Pneumothorax has effected a 75 per cent collapse. Mediastinal hernia on left side with general shift of mediastinum. Cavity much smaller.

The child was maintained on the usual sanitarium régime of an active tuberculous case. An *x*-ray taken six months later (January 14,

1935) again showed the remarkable tendency to clear and spread. (Fig. 6.) Resolution and fibrosis were starting to take place on the left side and the original focus on the right side revealed fibrosis and possibly an ill defined cavity. However, in the second anterior interspace, a new lesion was forming.

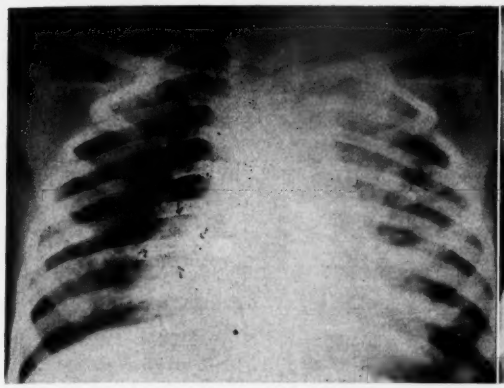

Fig. 5. July 18, 1934. Slight rise of right diaphragm following phrenicectomy. Fibrosis at site of original lesion. A new exudate spread of left upper lobe.

Fig. 6. January 4, 1935. Resolution and fibrosis taking place in left upper lobe. A new lesion is forming in second anterior interspace on right.

A few months afterward, he started to reveal definite signs in the right upper lobe and his sputum became more profuse. An extensive lesion of the right upper lobe with cavity formation was noted in an *x*-ray taken May 16. (Fig. 7.) The original lesion seemed to be undergoing fibrosis, while the left side continued to clear with fibrosis and calcific deposition as residue.

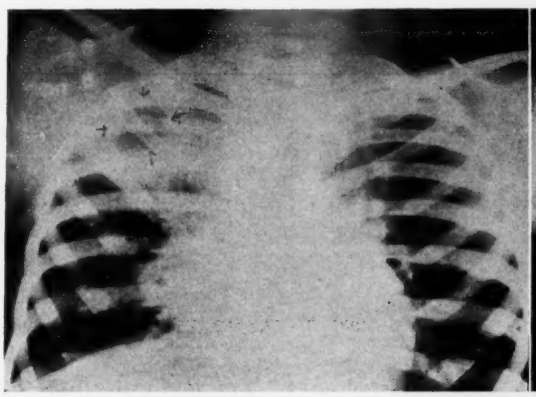

Fig. 7. May 16, 1935. New lesion in right upper lobe is becoming more extensive and cavitation taking place.

Fig. 8. May 29, 1936. Lesion still active in right upper lobe. Left side fairly clear.

The uncoöperative guardian now stepped into the picture and removed the child to her home, where he remained from May 29 to December 5, 1935. Since cough and expectoration continued and his general condition seemed slightly worse, he was sent back for further care.

On his readmission there was no essential change in laboratory work-up from his first admission to Essex County Isolation Hospital. An attempt was made to start pneumothorax again, but this met with persistent failure, for the pleura was now completely adherent. At this time thoracoplasty was suggested in spite of the child's age, since no alternative could be offered. We decided to observe the left side for several more months and then proceed with surgery.

The left side was considered satisfactory both from a clinical and a roentgenologic viewpoint on May 29. (Fig. 8.) A two-stage thoracoplasty was then performed by one of us (R. H. D.).

Technique and Course. First stage thoracoplasty was done June 22, 1936 under avertin, nitrous oxide and oxygen anesthesia. The first,

second and third ribs were resected, and the first removed to the costocartilage. Smaller sections than usual were removed from the

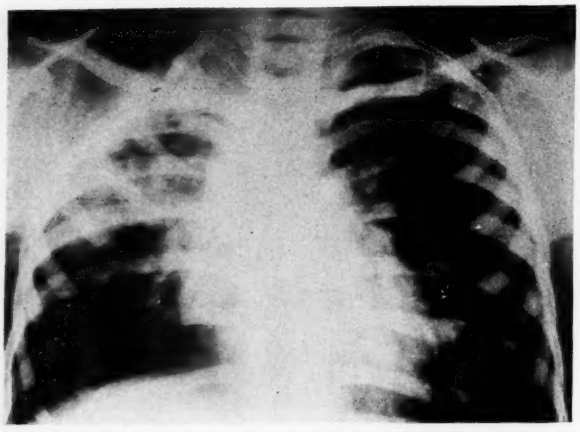

Fig. 9. October 1936. After first stage thoracoplasty. Three upper ribs removed.

second and third. This was done for a two-fold purpose: (1) it was thought than on account of the age of the patient, the bony cage would be more yielding and good collapse obtained with less sacrifice of support; and (2) we hoped that there would be less tendency to spinal deformity. The wound was closed in layers and a Penrose drain inserted. The duration of the operation was fifty minutes, the patient's condition remaining good

The postoperative course was uneventful, the highest temperature being 101. Slight serous drainage took place from the wound. The left side was relatively clear in October, after the first stage had been completed. (Fig. 10.)

On October 23, a second stage thoracoplasty was done under the same anesthesia as the first. The fourth, fifth and sixth ribs were resected, and a revision of the second and third was done. Closure was done without drainage. The patient stood the procedure well and convalesence was uneventful. The wound was clean. A Bucky roentgenogram, taken in February 1937, demonstrated collapse and beginning spinal deformity. (Fig. 10.)

In a child so young one would naturally expect some postoperative spinal deformity, and for this reason the resections were less extensive. A spinal fusion at the time of operation has been suggested and performed by Dr. Coryllos. Of late Dr. Coryllos has been using a rib graft,[1] and this would probably have obviated the spinal curvature, but at the time we were not familiar with the procedure. No attempt was made to free the lobe as in the Semb operation,[2] because in one so young it was felt that the mediastinal structures would be unduly yielding and too much of a shift obtained.

At present the patient is doing very well. He is maintained on a modified sanitarium régime with very little liberty. The fear of deformity of the spine has always been present and the orthopedic service is taking care of this phase of a special brace. His general condition has improved considerably, his appetite has increased and mentally he seems happier.

It is interesting to note that the sputum has diminished and is negligible in quantity. On the second admission to Essex County Isolation

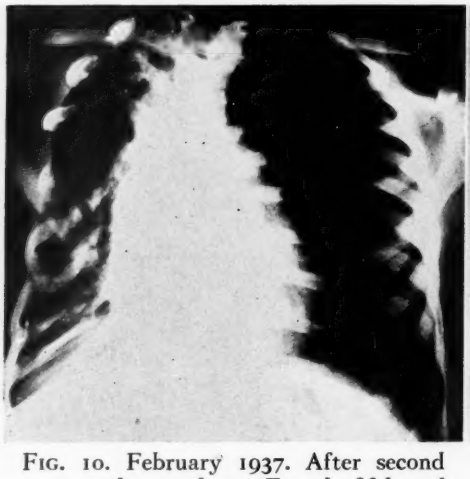

Fig. 10. February 1937. After second stage thoracoplasty. Fourth, fifth and sixth ribs removed. Bucky *x*-ray also reveals beginning spinal deformity.

Fig. 11. November 1936. Photograph of child taken one month after second stage thoracoplasty.

Hospital the sputum was positive and remained so fairly consistently until the first stage thoracoplasty. After this only an occasional positive was noted. Since the second stage thoracoplasty in October 1936, no positive sputum has been detected.

The sedimentation rate also is worthy of noted. The Westergren method, where a high normal is considered 25, was utilized. In the first thoracoplasty the rate was 83. After second stage thoracoplasty the rate was 67. In December 1936, the rate was 25, and in February 1937, the rate was 18.

The general trend of improvement in every direction, corroborated by laboratory data and roentgenograms is very gratifying. (Table I.)

TABLE I

GENERAL PICTURE 1933–1937

	Incomplete Pneumothorax 1933	Phrenicectomy 1934	1935	After First Thoracoplasty 1936	After Second Operation 1937
Sedimentation rate (Westergren)	.....	.....	83	67	18–25
Sputum Bacilli	Positive	Positive	Positive	Occasional positive	Negative
Sputum Amount	Abundant	Moderate	Moderate	Diminished	None
Temperature	99–101	99–101	99–100	99–100	98–99
Weight	39 pounds	41½ pounds	50 pounds	61 pounds	68 pounds
Toxic Symptoms	Present	Present	Present	Diminished	Absent

COMMENT

This child was quite young when artificial pneumothorax was instituted, but younger cases have been reported. Gardere and Wenger,[3] after a study covering over twenty years, believe that the procedure is beneficial in ulcerocaseous forms of tuberculosis in children, but admit only a 20 per cent satisfactory and lasting result. Gross and English[4] state that whenever artificial pneumothorax could be adequately employed, the results were far superior to any other type of compression. This is in accord with our opinion and it is therefore regrettable that refills could not be given. G. Zederbauer[5] states that avertin anesthesia may be given in very young or excitable children, but this was considered objectionable. The phrenicectomy which followed in this case was of little value.

This child, age 9, is the youngest instance of thoracoplasty in tuberculosis that we could uncover in the literature. Fishberg[6] states that Maurice Davis performed thoracoplasty for bronchiectasis in children 3½, 5 and 7 years old, and in one case of tuberculosis in a child of 12 years. He was unable to encounter one instance of operation being performed for tuberculosis in a child under 12 years of age. According to Alexander,[7] thoracoplasty should be limited to patients between the ages of 15 and 45. In 1913, Sauerbruch[8] cited Lenhartz as performing a complete operation in a child of 12. Jacobaeus and Key[9] report bad results in patients less than 20 years of age and state that surgeons are hesitant to perform thoracoplasty, but they believe that some children should have the benefit of this procedure.

The fact that the collapse effected by the thoracoplasty is not as great as usually desired, is appreciated. It is realized that we were too cautious and conservative, but the opportunity to perform this type of surgery in one so young is rare. It is suggested that in the future a greater portion of each rib may be removed. The follow-up of this case is rather brief (ten months) and therefore, no conclusions can be definitely outlined. The possibility of reactivation and future revision procedures is not

unlikely. Despite the several unfavorable authoritative opinions voiced on the eventual outcome of this case, we offer it as a preliminary report and hope to review its progress in the next few years.

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Regeneration does occur with certain human tissue and under certain circumstances. The liver, for example, regenerates perfectly even when almost all of it has been removed, and so do some other parenchymatous organs, but to a lesser extent.

NON-CASEOUS TUBERCULOSIS OF THE THYROID GLAND

REPORT OF A CASE

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TUBERCULOSIS of the thyroid gland, though rare, occurs more frequently than was once thought. Careful microscopic study of resected thyroid tissue probably accounts for the apparent increase in incidence.

Rankin and Graham reported a group of cases several years ago, a large percentage of which presented symptoms of hyperthyroidism and were found to have high basal metabolic rates. Microscopic examination of the gland revealed the structure typical of exophthalmic goiter associated with the tuberculous involvement in many cases. Usually tubercles and giant cells were seen. A few cases of tuberculous thyroiditis unassociated with adenomas and hypertrophic changes were found. No pulmonary tuberculosis was found in any of these cases, and the original focus could not be determined; the authors concluded that it was too small to find. However, it seemed logical that the thyroid gland was invaded secondarily, probably as a result of tuberculous bacillemia. Van Ravenswaay and van Ravenswaay concur with Rankin and Graham that a diagnosis can be made from the microscopic picture with a high degree of accuracy. Most writers disagree with Cohen who stated that the diagnosis should be made clinically.

CASE REPORT

A 60 year old white woman was seen July 29, 1937. She complained of a lump in the right side of her neck, which she had first noticed about six months before. It had gradually increased in size since that time.

She had been hoarse since 1904, but the degree of hoarseness varied from time to time. She had always been thin and had frequent colds with a great deal of sputum. A pelvic tumor was removed in 1916. She had been told that she had pulmonary tuberculosis in 1920, but had taken no treatment. In 1923 another examination revealed "pulmonary tuberculosis with many cavities" and "heart trouble." However, she went on teaching school.

She had been told that the deaths of two of her sisters, each at the age of 23 years, had been due to tuberculosis, but she had not lived with them for several years prior to their deaths.

Examination revealed the patient to be underweight by about 20 pounds. Her temperature was 98.6; pulse 100; blood pressure 150/100. There were no eye signs indicative of exophthalmic goiter. The larynx and vocal cords were normal. The right lobe of the thyroid gland was enlarged and very hard, and seemed to be attached to the thyroid cartilage. The heart was normal and there were harsh breath sounds present over both apices. The abdomen was negative except for a low midline scar. Pelvic examination was essentially negative. *X*-ray of the chest showed, old fibrotic changes in both apices and at the left costophrenic angle. No areas of soft tissue infiltration suggestive of active tuberculosis were detected in the lung fields. The density of the lung fields suggested an emphysematous condition. Blood studies showed hemoglobin 89 per cent, 4,120,000 red blood cells, 4,850 white blood cells, 78 per cent polmorphonuclear leukocytes, 19 per cent small lymphocytes, 3 per cent large lymphocytes. An uncatheterized urine specimen contained an occasional pus cell. The basal metabolic rate was minus 9.

A diagnosis of adenomatous goiter without hyperthyroidism was made. It was felt that the adenoma had probably become malignant.

On August 2, under local and gas anesthesia, the entire right lobe, isthmus and one-half of the left lobe were resected. The right lobe was very adherent to the muscles and trachea. The

FIG. 1. Section of thyroid gland showing non-caseous tuberculosis. (High power.)

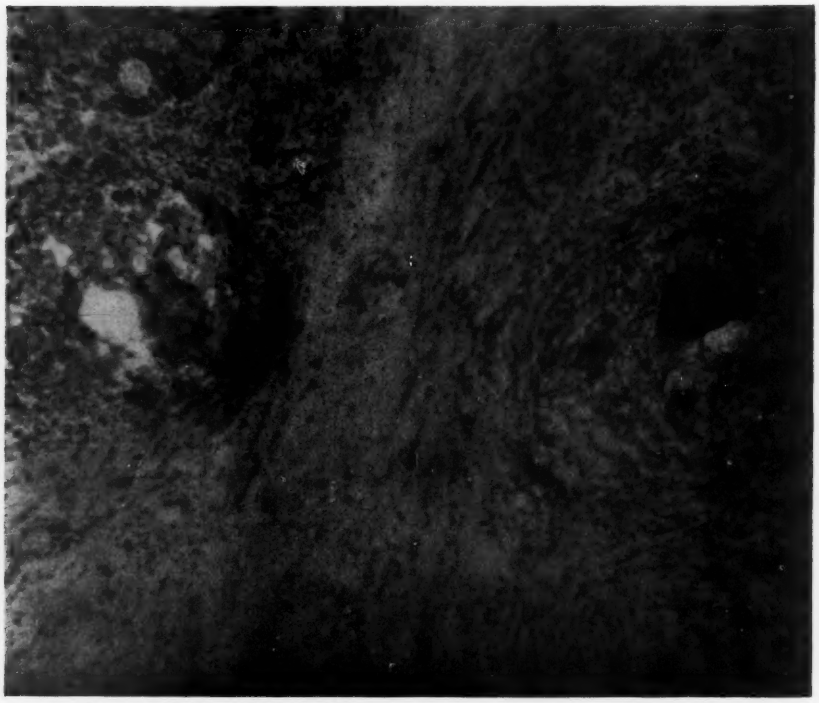

FIG. 2. Section of thyroid gland showing non-caseous tuberculosis. (Low power.)

resected thyroid tissue measured 5 × 4 × 3 cm. and grossly was grayish-white in color and very firm in consistency. The microscopic sections showed a diffuse non-caseous tuberculosis.

The immediate postoperative course was uneventful. The wound healed promptly, but the hoarseness has persisted as before though there is normal movement of the vocal cords.

This case is of unusual interest in that it gives a more definite clue to the primary site of infection than most others reported. Two and one-half months following operation symptoms of a mild hypothyroidism developed and the basal metabolic rate was found to be minus 16. Administration of desiccated thyroid gr. 1 daily brought the rate up to normal and since that time ½ gr. daily suffices. The patient feels well and carries on her regular teaching duties.

CONCLUSIONS

1. Tuberculosis of the thyroid is rare.
2. Most cases have associated hyperthyroidism.
3. The diagnosis is almost never made clinically.
4. The prognosis is usually good.
5. The case presented is of unusual interest in that it gives a more definite clue to the primary site of infection than most others reported. The pulmonary tuberculosis has apparently healed. There has never been any evidence of laryngeal tuberculosis. There was no hyperthyroidism. The wound healed readily without prolonged drainage. With the exception of a mild hypofunction of the remaining portion of thyroid tissue, which is easily controlled by the administration of desiccated thyroid, the patient is in good health.

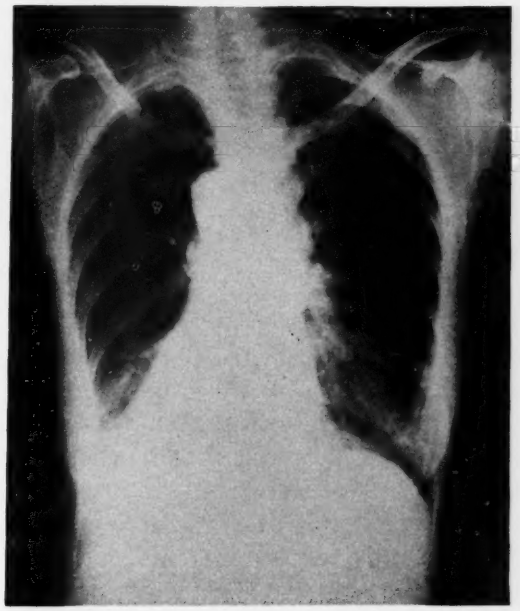

Fig. 3. Recent roentgenogram of chest showing fibrosis in both bases and no evidence of active tuberculosis in the apices.

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THREE CASES OF CONGENITAL UMBILICAL HERNIA*

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TO find the cause of congenital umbilical hernia we have to go back to the early weeks of embryonic life. At this time the primitive alimentary tract is divided into three main subdivisions—the foregut, the midgut and the hindgut. At about the fifth week of life the midgut is pushed out through the umbilicus and there is a physiologic umbilical hernia, due to the rapid increase in the length of the midgut and the marked increase in the size of the liver. The growth of these two organs is out of proportion to the growth of the rest of the abdomen, and the abdominal cavity is not large enough for all the structures. Between the second and third month of embryonic life it is perfectly normal for this midgut loop to develop within the umbilical sac.

About the tenth week of life the alimentary tract begins to go through the second stage of rotation, and this traction on the loops of bowel usually results in the return of the midgut section of the abdomen. Failure of withdrawal at this time is now generally thought to be the primary cause of congenital umbilical hernia. The intervention of these organs which should have receded into the abdominal cavity prevents the proper approximation of the abdominal muscles on the two sides at the midline. Instead of the umbilical cord emerging from a small opening there is really no umbilicus—only a large oval-shaped defect in the midpart of the abdominal wall through which the viscera protrude into the umbilical cord. The protruding mass may be large or small intestine or both. The liver is frequently within the mass and the spleen may also be within it.

Incidence. Congenital umbilical hernia is an extremely rare finding. According to Jarcho, not more than 356 cases have been reported in the literature. He points out that there must have been many cases in stillborn infants or in those who died shortly after birth, which have never been reported.

Diagnosis. The diagnosis presents no difficulty. It is self-evident as soon as the child is delivered. The hernial sac is often translucent and the contained viscera can easily be seen through it.

CASE 1. A baby girl was born August 25, 1935 after a normal labor. Delivery was spontaneous. The child weighed 6 pounds 2 ounces. There was a tense mass the size of a baseball in the umbilical region, covered by a thin serous membrane through which could be seen loops of intestine. It was impossible to reduce the mass and it was easy to see that any great amount of pressure would rupture the covering membrane.

Operation was performed just three hours after birth under chloroform-ether and oxygen anesthesia. The sac was opened and found to contain about one-half the small intestine, the appendix, the ascending colon, and part of the transverse colon. The loops of bowel, which were adherent to the sac, were freed by sharp dissection and returned to the abdomen. There was some difficulty in this, as the abdominal cavity was not used to containing such a large mass of organs. The sac was trimmed flush to the edges of the abdominal wall and all vessels were ligated. No attempt was made to separate the layers of the abdominal wall. The abdomen was closed with interrupted through and through black silk sutures. The time of operation was twenty-two minutes.

A clysis of 75 c.c. of normal saline was given. The temperature that evening was 104.8, but came down the next day and remained down.

* From the surgical service of Muhlenberg Hospital, Plainfield, New Jersey.

The wound healed by primary union. The baby was discharged twenty-five days after operation, at which time she weighed 6 pounds and 15 ounces—a gain of 13 ounces over the birth weight.

In spite of the uneventful postoperative course the child showed certain signs of mongolism. There was a heavy pad of fat at the base of the neck, the fontanelles did not close on time, and the child was very susceptible to every infection. It died at four months of age of lobar pneumonia. The father and mother were both normal and healthy. There were two other children, 11 and 14 years of age, both normal and healthy.

Case II. This male infant was born September 15, 1935. Labor was normal and delivery spontaneous. Weight was 7 pounds and 4 ounces. In this case there was a large, dark-colored, oval-shaped mass in the umbilical region. Our belief that it contained liver proved to be correct.

The baby was operated on a little less than three hours after birth. A few drops of chloroform were used. The lower part of the sac was opened and the contents explored. There was no bowel in the sac but the whole anterior half of the liver was firmly adherent to its upper part. It was just as if the peritoneum covering the anterior surface of the liver were directly continuous with the skin on either side of the abdomen. We saw that it would be impossible to free the sac from the anterior surface of the liver—it would be just like trying to dissect the peritoneum off the liver, since this one layer of tissue was in reality both the peritoneal covering of the liver and the outer or skin covering of the abdominal wall at this point. We trimmed the sac away from the edges of the abdominal wall on either side and then cut it away from the liver all the way around at the point where the sac became adherent to the liver. We had to leave the center of the sac permanently attached to the anterior surface of the liver. We then returned the liver to the abdomen and approximated the two sides of the abdominal wall with interrupted through and through black silk sutures. The defect in the abdominal wall was about 4 inches in length. The time of operation was twenty-one minutes.

Our great fear in this case was that there would be peritonitis from the anterior surface of the liver which had served as a skin covering to this part of the abdominal wall. However, nothing happened. We gave the baby 40 c.c. of his father's blood. He made an uneventful recovery and was discharged on the fourteenth

Fig. 1. Case III.

day, when his weight was 7 pounds and 10 ounces, a gain of 6 ounces over the birth weight. I saw the child again when he was a little over 2 years of age and weighed 24 pounds. He is healthy, active, and intelligent, but I do not think he is quite as sturdy as the other children in this family. Both parents are normal and healthy. There are four other children, one born after this baby. All are normal.

Case III. Baby girl, born February 11, 1938, was a first child and full term. The labor was normal and the delivery spontaneous. The weight was 6 pounds and 3 ounces. In this case there was a tense mass the size of a small grapefruit occupying the center portion of the abdomen and covered by a thin membrane through which could be seen the liver and the coils of intestine. The cord was tied off to the left side of the sac.

The baby was operated on one and one-half hours after birth under chloroform, ether, and oxygen anesthesia. The sac was opened and explored. It contained the liver, spleen, ascend-

ing colon, transverse colon, and the small intestine. Almost all the viscera with the exception of the stomach and the descending colon were out of the abdomen and in the sac. There were many fine adhesions between the sac and contents, but these were freed easily by finger dissection. The sac was then trimmed away all around at the skin margin.

It was very evident at the start that it would be a most difficult, if not impossible, task to return all these organs to the abdomen, since the abdominal cavity had not developed in size with the organs. The fact that these organs had been outside the abdomen for so many months meant that there had not been sufficient stimulation for the proper enlargement of the abdomen. Their return required considerable manipulation and much unavoidable pressure. The wound was closed with through and through black silk retention sutures. The lower part of the wound was rather tense and it was evident that there was considerable pressure on the contained viscera. It seemed rather doubtful that they could adjust themselves to this pressure.

The baby was given 40 c.c. of father's blood in the muscles and this was repeated on subsequent days. Clyses of normal saline with 5 per cent glucose were also given at frequent intervals. A karo mixture was given by mouth, but very little of this was retained. There was some vomiting of dark bloody material from the start. The baby had two to three bowel movements every day and these appeared to contain blood. The rapid respiration and cyanosis which persisted from the time of operation were very striking. There was apparently tremendous pressure exerted against the diaphragm. The temperature was 98.6 the day following operation, but rose to 102.4 twenty-four hours later and remained up. It was evident from the beginning that this baby would not survive. The vomiting was frequent, the respirations fast, and the cyanosis marked. The baby died three days following operation. Autopsy was refused.

Treatment. Every case of congenital umbilical hernia should be operated on immediately, provided surgical means are available. The sac should be incised, the contents freed from the sac and reduced, and the abdominal wall closed. The operation should be done at once even though the contents of the sac can be reduced. Delay results in desiccation of the sac and the possibility of its being torn. If desiccation results, it is almost sure to be followed by infection and peritonitis.

CONCLUSION

The three cases presented here demonstrate that the operation for congenital umbilical hernia may be either a relatively simple procedure or an extremely difficult one with little chance of success. It all depends on how much of the gastrointestinal tract is in the sac and has to be returned to the abdomen. In any event, the earlier the operation is performed the greater are the changes of success. Babies seem to stand the operative shock very well.

PARAFFINOMA OF MALE BREAST*

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THE injection of melted paraffin to correct disfigurements, defects, impaired physiologic function and certain other pathologic conditions seemed to be a relatively simple and satisfactory operation when first advocated thirty-eight years ago. At that time Gersuny recommended the injection of paraffin of low melting point to correct depressed scars, and the method was received with enthusiasm. Pure paraffin ($C_{27}H_{56}$) was thought to be a harmless and indifferent material which would be tolerated by the body organism and afford permanent results. Gersuny also recommended it as a means of correcting certain speech defects by injecting the paraffin into the soft palate and uvula. At the time it appeared to be an effective procedure, as, indeed, it proved to be one of the simplest.

With happy optimism, saddle nose, pendulous breasts and various facial defects were corrected with the pliable paraffin to the utmost satisfaction of the patient.

The injections of paraffin at low melting point—40°C. (104°F.)—were used not only as an aid to cosmetic surgery, to correct depressions and wrinkles, but were also recommended in the character of prothesis. Surgeons claimed excellent results in cases of urinary incontinence, anal prolapse and even hernia. In ozena, paraffin was used as a trophic stimulant. Voice defects were corrected in paralysis of the vocal cords by paraffin injection. It was even used in abdominal surgery to prevent adhesions.

But reports of complications began to appear in the literature. Emboli occurring after paraffin injections brought the practice into disrepute. Kofman was unfortunate enough to see one of his patients die of pulmonary embolism after a cosmetic operation. Complaints were made that the injected paraffin became displaced, producing additional disfigurements and necessitating operative intervention for its removal. In certain instances paraffin injected into the cheeks migrated downward over the jaw and produced serious disfigurement (Sehrt; Wassermann).

Eckstein claimed that emboli could be prevented by injecting paraffin of a higher melting point (58°C.) and believed that these complications were merely due to the use of paraffin of low melting point. His recommendation was gladly accepted and the technique was modified by the use of a high pressure injection machine. This method was largely practiced in the field of cosmetic surgery until numerous late complications were reported.

Sehrt observed a case in which surgery was necessary to remove the paraffin together with the adjacent chronically inflamed tissue, and Benedek demonstrated that paraffin injected into a patient's breasts gave rise to a chronic productive inflammation thirteen years later. He called the condition paraffinoma. In this case both breasts, which had previously been injected for cosmetic purposes, were operated upon because the condition simulated malignant disease. Paraffinomas of the breast developing from two to twenty years after the injections were reported by others. In Rose's case both breasts had to be amputated eighteen years after paraffin injections had been used for cosmetic purposes.

During the World War malingerers had paraffin injected into their tissues so as to create artificial tumors which would enable them to escape military service. Flandin

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observed a paraffinoma of the instep following an injection nineteen years earlier. Fischer-Wassel described a case of fatal

Fig. 1. Ulcerated paraffinoma of the male breast.

cirrhosis of the lung after protracted use of nasal menthol oil applications. Jaffa reported a case in which the administration of paraffin oil by mouth might have been the cause of an appendicitis. It is, of course, questionable whether the paraffin found in the appendix was the cause of the appendicitis or whether it had lodged in the appendix because of that organ's pathologic condition.

Whittaker observed ulceration in the bladder following paraffin injection for hernia.

During the period when oleothorax was used, numerous observations of paraffinomas of the thoracic wall were described.

A few typical adenocarcinomas were found in breast previously subjected to paraffin injections for cosmetic purposes. Despite this finding there is no proof (though a constant chronic irritation might be considered the underlying cause of the development of the cancer) that the paraffin is the cause of the cancer, and in view of the relative frequency of cancer of the breast it may be regarded as mere coincidence.

Pathologically, paraffin injections result in foreign body granulomas with no tendency to malignancy. Bolam, Weidman, Jaffa and others observed that such paraffin forms a localized tumor and, as Baer pointed out, the deformities may develop within six months to five years later. It is of more than passing interest that the injection of vegetable oils shows no tendency to tumor formation. Paraffin acts as a foreign body and the resulting irritation produces a benign granuloma which is not a true neoplasm (Weidman). Flandin and others have claimed success with *x*-ray treatment of such small granulomas beneath the eyelid, thus obviating surgical interference. There was, however, no proper follow-up of the case reported. In our own case which is unique in the available literature we have observed this as a wandering of the paraffin to the regional lymph nodes accompanied by secondary inflammation. No lymph node involvement was noted by other reports.

CASE REPORT*

W. S., an actor, a white male of 34, and single, was referred to our clinic from Florida with the diagnosis of possible cancer of the right breast.

He stated that four years previously he had been troubled with "contractions and a throbbing sensation" in his right breast for which condition he received various forms of physiotherapy, including *x*-ray, in Paris, and that about two years before, a physician there had injected some kind of fluid into the breast without effect. About two months before examination the patient noticed that an area in the right breast was becoming red and slightly painful. This opened and discharged some serous fluid. About this time he noticed a lump in the right axilla and consulted his physician in Miami who advised him to come to our clinic. The family history was irrelevant.

The face was asymmetrical. The right side was drawn upward, showing definite contraction over the mid-portion of the right mandible. Numerous small dilated blood vessels were present in this region. The right side of the neck extending downward to the chest and into the upper and lower arm on the outside surface presented telangiectasia, probably as the result of previous *x*-ray treatments.

* For analysis of male breast tumors see Cholnoky, Tibor de. Benign fibrous tumors of the male breast. *Am. J. Surg.*, 30: 298; 309; 396, 1935. Benign tumors of the breast. *Arch. Surg.*, 38: 79–98, 1939.

The right breast was involved by an ulcerative process approximately 2 inches in diameter which had entirely destroyed the nipple and was surrounded by a bluish red area extending over the whole breast toward the sternum and axilla. The mass was rather firm and there were stony hard infiltrations over the surrounding inflamed areas. The entire lesion was slightly tender and unattached to the underlying tissues, that is, it did not seem to be adherent to the pectoral fascia or muscle. There were several large and slightly tender lymph nodes in the right axilla. The other breast was normal.

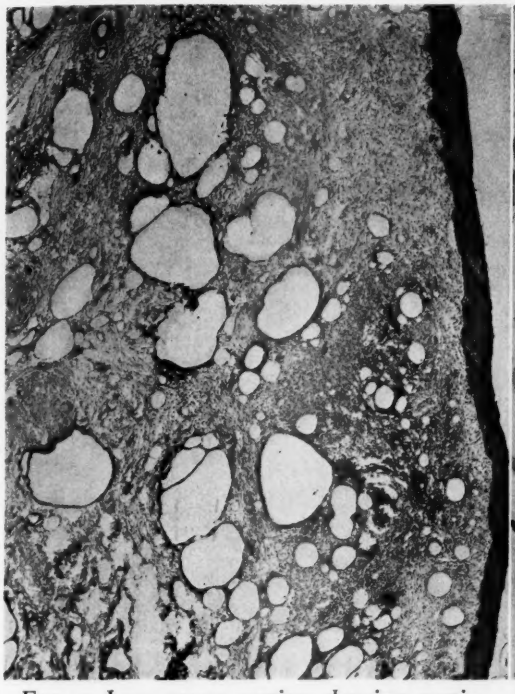

Fig. 2. Low power section showing cystic spaces in derma with inflammatory infiltration of surrounding stroma.

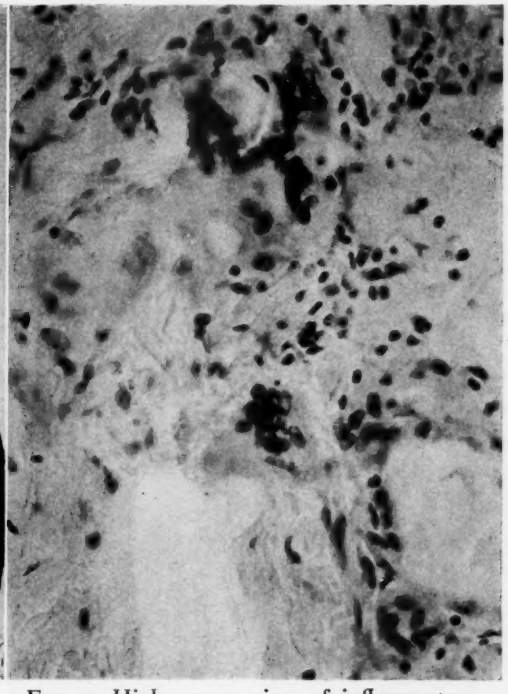

Fig. 3. High power view of inflammatory changes in stroma showing foreign body giant cells. The cystic spaces show no epithelial lining.

The Wassermann reaction was negative. Urinalysis was negative. The blood showed a mild secondary anemia.

A clinical diagnosis of ulcerated tumor, possibly carcinoma of the right breast was made, but the possibility of a chronic inflammatory condition caused by previous paraffin injections had to be kept in mind. The patient was advised to enter the hospital for an operation, which was performed March 5, 1932. A biopsy specimen was secured with an electric loop. During this procedure a soft white material, recognized as paraffin, ran out of the wound. Frozen section confirmed the diagnosis of a chronic inflammatory mastitis and a diagnosis of paraffinoma was made. The indurated and ulcerated inflammatory areas were circumscribed by the electric cutting blade and resected en masse. Pieces of paraffin vaying from 1 mm. to 1 cm. in diameter were found distributed in the subcutaneous fatty tissues. All indurated fatty tissue between the skin and pectoral fascia was removed. The skin and pectoral fascia did not seem to be involved in the process. The enlarged axillary nodes were carefully removed. After hemostasis a rubber drain was inserted and the skin was united with interrupted black silk sutures. The wound healed normally.

Pathologic examination was done by Dr. D. S. D. Jessup, pathologist of the tumor clinic, and the report may be quoted as follows: "The section shows fibrous tissue with enlarged cystic glands and inflammatory infiltration giving the appearance of a cystic mastitis without epithelial lining of the cystic spaces. There are foreign body giant cells in the stroma with some areas of necrosis. These fibrous cystic changes are characteristic of paraffinoma."

The patient returned four weeks later with an anal fistula which was operated upon, and meanwhile a red and definitely tender mass about one inch in diameter appeared in the

right axilla. Fluctuation soon developed; the mass was opened and drained and about 5 c.c. of seropurulent fluid was obtained. Healing occurred by secondary granulation after two weeks during which there was a fair amount of daily discharge.

Subsequent neurologic examination was done by Dr. Byron Stookey who diagnosed an atrophy of the facial muscles as a probable result of a discrete lesion of the nerve nucleus.

COMMENT

After paraffin is injected under the skin it remains soft and pliable, usually for a number of years, but then it gradually hardens and visible changes occur in the tissues.

Paraffin ($C_{27}H_{56}$) in its chemically pure form is not directly toxic to the person using it, but acts as a foreign body. It irritates the surrounding tissues so that connective tissue strands grow into it and the enmeshed paraffin becomes a hard tumor. This chronic inflammatory condition in some cases may develop to the point of ulceration, and after the skin has broken down the paraffin may exude. Although the paraffin is likely to cause such pathologic changes, it cannot be stated that all injected cases exhibit such late complications, although Mook and Wander stated that a single injection of paraffin may lead to the production of tumors. Considering the relatively small number of paraffinomas one cannot assume that such tumors develop in every case although we have no definite proof such as would be furnished by a follow-up on all injected cases. It might be supposed on the other hand that paraffinomas develop earlier in individuals who are more susceptible to the irritation caused by the paraffin injections and in the majority of such cases the tumors appear five to twenty years later. These disfiguring tumors often have to be excised together with all the paraffin-bearing tissues to prevent recurrences. This may necessitate complicated plastic reconstruction.

The actual reason for the paraffin injection in our patient is not clear. According to him it was done in an effort to treat his nervous condition. A more likely explanation might be that the alleged *x*-ray treatments were employed to produce epilation on the right side of the chest and face, which in addition to an artificially tumified breast, would produce a feminine appearance of one-half of the body and permit him to earn a living as a side-show freak.

Paraffin injections should be condemned as harmful to the patient, causing late complications which often require plastic surgical operations and transplantations of autogenous tissues to correct the malformations and deformities so caused. In the plastic correction of disfigurements and defects autogenous material, for example, fatty tissue, should be used.

SUMMARY

One case is described of paraffinoma of the male breast which developed two years after the injection of paraffin for an undetermined purpose and in which there is a four year follow-up. No definite conclusion can be reached as to whether paraffin injections cause malignancies, although the tumor called paraffinoma (foreign body granuloma) might follow many months or years after the injection of paraffin of lower (40°C.) and higher (58°C.) melting point.

The treatment of choice is a thorough extirpation of the paraffin-bearing tissues, followed, if necessary, by plastic repair.

In plastic surgery the use of autogenous material is recommended.

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Once an infection has gained a foothold, it is obvious that a few million dead bacteria [in a vaccine] cannot stimulate the production of immune bodies as well or as rapidly as the many live organisms already invading the body.

STOMA STRAIN FOLLOWING GASTRIC SURGERY

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A REVIEW of the literature reveals no mention of stoma strain following gastric surgery. Ptosis of the abdominal viscera has been discussed at length, but not in association with the various operations performed for ulcer or carcinoma.

In the three cases which are presented, the main complaints were pain, weakness, vomiting, fatigue, and the inability to carry on work, especially when the patient was standing for any length of time. These patients were tall, thin and long-waisted, with relaxed abdominal walls. All three were operated on for peptic ulcer, Case II having been operated upon twice, but the weakness, fatigue and ulcer syndrome persisted. The recurrence of the ulcer syndrome was aided by the indiscretions of the patients, all of whom used tobacco and alcohol in more than moderate quantities. The pain, vomiting and heartburn of the ulcer syndrome cleared up with rest, alkalis, diet and sedation, but the weakness and fatigue persisted. The fact that the operations had been performed by excellent surgeons led me to consider other factors which would give relief to these patients.

A consideration of the supports of the stomach reveals that the fixed supports of this organ are at the esophagus and the pylorus. The other supports are peritoneal attachments forming so-called ligaments along the lesser curvature (the gastrohepatic) and the greater curvature (the gastrocolic and the gastrosplenic). However, between the esophagus and the pylorus the stomach is simply a flexible, movable bag. In gastric surgery, the stomach is sutured most often to the jejunum, another flexible, movable organ.

Try as the surgeon may, he cannot support the stoma resulting from the operation as the stoma at the pylorus and esophagus are supported. Hence, there is a downward pull by the small intestines and the transverse colon which causes added strain on the stoma. To this factor, add the long-waisted, thin, relaxed abdominal wall which predisposes to ptosis of the abdominal viscera, and the strain on the surgical stoma is undoubtedly increased.

In the following cases, relief has been afforded the patients by the use of a hypogastric supporter, a pad with the convex surface pressing against the lower abdomen, giving support to the abdominal viscera and thereby lessening the strain on the stoma. The relief from strain may also improve the circulation about the stoma, and this would be a factor in preventing the occurrence of marginal ulcer.

CASE I. W. McK., age 67, an electrician, was first seen on March 31, 1936. He had been operated upon at St. Luke's Hospital in New York City about fifteen years previously for stomach ulcers. *X*-ray revealed a partial gastrectomy.

The patient complained of weakness and fatigue after working a few hours on his feet. He had nausea and vomiting on taking solid food only, and suffered from epigastric pain, relieved by milk and alkalies. Heartburn and loss of weight were further symptoms. This man was a heavy user of alcohol and tobacco. Beyond the stomach complaint he had always been well.

He was a tall, long-waisted individual with a thin, relaxed abdominal wall. His appearance was pale and drawn, he was bent over, and spoke as though extremely tired. The heart tones were dull and there was sinus rhythm. The blood pressure was 150/80, pulse 80,

hemoglobin 70 per cent (Tallquist). Clinically he appeared arteriosclerotic.

Electrocardiograph findings were myocardial damage, without coronary damage.

the small intestine with considerable pull on the stoma, partially occluding it. When the intestinal loops were filled, the pain and nausea began. Medication gave no relief.

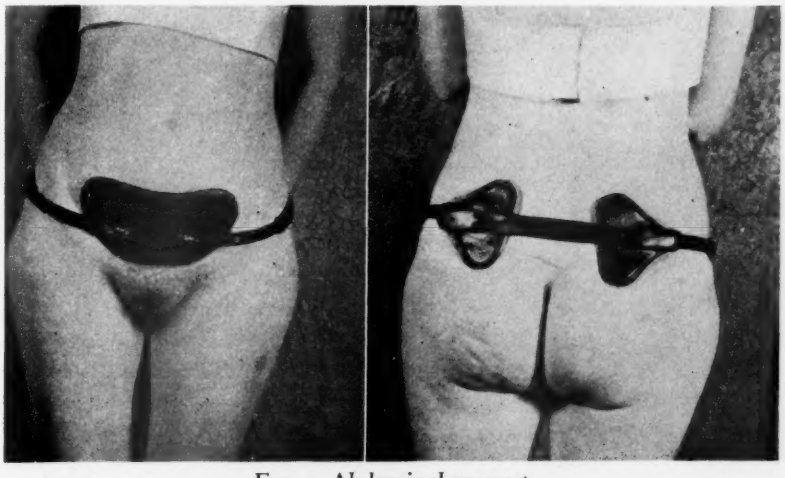

Fig. 1. Abdominal support.

At this time, the diagnosis was one of myocardial damage, with peptic ulcer (marginal), and malnutrition. *X*-ray revealed an ulcer at the stoma, with no obstruction present. Treatment consisted of twenty-four injections of larostidin, modified sippy diet, and alkali therapy. Within three weeks the pain, nausea and heartburn had subsided. However, the weakness and the fatigue persisted and no gain in weight occurred. The marked ptosis of the small and large bowel was considered to be causing the latter symptoms. Hypogastric support was given, with the result that within two months there was a gradual subsidence of the fatigue and weakness, together with a gain in weight of 20 pounds. In January 1937 there was a slight recurrence of the ulcer syndrome following excessive beer drinking but there was no recurrence of the weakness and fatigue.

Case II. D. O'C., age 38(?), a policeman, was examined October 22, 1933. He had had a simple gastroenterostomy done at the Lenox Hill Hospital (New York) and a gastric resection at the Mayo Clinic.

He complained chiefly of weakness and fatigue after a few hours in the erect position, epigastric pain, and vomiting (solids or liquids), except when in the prone position. He was a habitual smoker and drank much beer.

This patient was also of the tall, long-waisted type, with thin, relaxed abdominal wall. There was no pathology evident on thorough examination. Fluoroscopy revealed marked ptosis of

Hypogastric support was given, which gave him relief from the pain and vomiting. In two months he gained 20 pounds, and the weakness and fatigue gradually disappeared. Occasionally he suffers acute epigastric distress because of his refusal to give up beer.

Case III. L. C., age 36, had had a simple gastroentorostomy done at Jefferson Hospital in 1925. This patient complained chiefly of occasional epigastric pain, weakness and fatigue after a few hours of standing on his feet, and occasional heartburn. Heavy use of tobacco and moderate use of alcohol were noted.

The patient was tall, with thin, relaxed abdominal wall, long-waisted in type. *X*-ray was negative for ulcer. The weakness, fatigue and the inability to gain weight were the principal complaints.

Treatment consisted of application of a hypogastric supporter. In six weeks there was a marked diminution in the weakness and the fatigue, together with a gain of 8 pounds.

The results obtained in the above cases justify a consideration of this appliance in the postoperative care of gastric surgery cases for the body type mentioned. The appliance is simply a metal pad with a convex surface covered with chamois and held in place with two flexible steel bands covered with leather and easily adjusted in the sacroiliac region. The belt is applied

in the prone position with the pelvis slightly elevated.

SUMMARY

1. Stoma strain is evidenced by weakness, fatigue and the inability to gain weight.

2. The stomas in these cases were functioning, except in Case II where the pull on the full jejunum partially occluded the stoma.

3. Most chronic ulcer patients are long-waisted and present a thin abdominal musculature.

4. Hypogastric support of the ptosed abdominal viscera relieved the symptoms of stoma strain.

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The canal of Nuck in the female corresponds to the processus vaginalis in the male and may be the source of herniation of the oblique (indirect) type of hernia.

CASE OF POSTTRAUMATIC STREPTOCOCCIC MENINGITIS SUCCESSFULLY TREATED WITH SULFANILAMIDE*

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THE value of sulfanilamide and related substances in the treatment of hemolytic streptococcal infections has been so well established that its application was considered as indicated in a case of a posttraumatic streptococcic meningitis.

REPORT OF CASE

For the sake of brevity only essential findings are recorded.

M. W., a man 52 years of age, was injured in an automobile accident and was admitted to the St. Elizabeth Hospital January 23, 1937 in a comatous condition.

Examination revealed a black discoloration of both eyelids on the right side, extensive suffusions and crepitation in the right frontal region, multiple bruises and lacerations of the face, epistaxis and broken upper central incisor teeth. The pupils reacted normally to light and accommodation; they were round and equal; no good view of the eyegrounds could be obtained. The patient vomited repeatedly. His blood pressure was 126/80; his pulse 60, and of poor quality; respiration 16, shallow; tendon reflexes normal. The roentgenogram of the skull showed a very extensive, depressed, comminuted fracture of the right frontal bone, extending through the anterior wall of the frontal sinus, the roof of the right orbit and into the base of the anterior fossa. In addition, according to the *x*-ray report, there was some apparent injury to the other facial bones, including the nasal bone, and an involvement of other nasal sinuses.

A diagnosis of basal skull fracture was made. The head of the bed was elevated, an ice bag applied to the frontal region and external heat to the extremities. Mild silver protein was instilled into the nose and tetanus antitoxin was administered. The fluid intake was limited. Intravenous injections of 300 c.c. of 50 per cent sucrose and hypodermic injections of caffeine sodium benzoate were given.

Two days after the accident the patient developed a typical delirium tremens. Micturition and defecation were involuntary. The agitation was successfully combated with paraldehyde enemas; no whiskey was given.

On the following day projectile vomiting developed; the patellar, triceps and biceps reflexes were absent, the Brudzinski and Kernig positive, the neck rigid. The reaction of the pupils could not be tested on account of the restlessness of the patient.

On January 30 a spinal puncture was performed, and the fluid was found to be cloudy, but under normal pressure. There was a cell count of 350 per c.mm., and Streptococcus hemolyticus was cultured. The patient was given subcutaneous injections of prontosil soluble; the total amount was 120 c.c. the first day and 90 c.c. the following days, divided into six doses daily. The following few days a marked subjective and objective improvement was noticed; the pathologic reflexes were absent; normal tendon and pupil reflexes reappeared.

On February 4 another spinal puncture was performed; the fluid was slightly cloudy, and there were 600 cells per c.mm. Culture showed no growth.

The temperature, which reached 103 degrees two days after the accident, fluctuated between 98 and 100.4 after the prontosil treatment was started. The injections were continued for six days. The patient recovered. Two months after the accident he was complaining of frequent headaches, but there were no other subjective symptoms.

COMMENT

Recovery from streptococcic meningitis is rare. Gray,[1] in presenting a sum-

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mary of the literature on this subject, reported sixty-six cases, including his own; he believes that the condition is at least 97 per cent fatal. Since his report there have been sixteen cases of recovery reported. Caussée, Loiseau and Gisselbrecht,[2] Schwentker and his collaborators,[3] Anderson,[4] Weinberg, Mellon and Shinn,[5] reported cases with recovery after the use of sulfanilamide and prontosil. However, in none of the cases did the meningitis follow an accident; usually it was of otogenic origin. The achievement of the drug in the case here reported is much more remarkable, as the meningitis was complicated by a skull fracture and delirium tremens, both factors contributing to the seriousness of the situation. It might therefore be advisable to test the *prophylactic* action of sulfanilamide in skull fractures if the presence of streptococci is demonstrated.

A noteworthy observation was the increase of the cell count in the spinal fluid two days after the injections were started; while before the treatment the count was 350 cells, two days later it rose to 600. Sulfanilamide is known to possess bacteriostatic properties; the above mentioned effect points to an increase of phagocytosis by the drug, as all other medication was discontinued as soon as prontosil injections were begun.

SUMMARY

1. A patient with a skull fracture complicated by delirium tremens and streptococcic meningitis was treated successfully with prontosil soluble.

2. To the author's knowledge, this is the first case of *post-traumatic* streptococcic meningitis successfully treated with sulfanilamide.

3. It is urged that the drug be used promptly in cases of post-traumatic streptococcic meningitis.

4. A suggestion is made to test the prophylactic value of the drug in skull fractures after the presence of streptococci has been demonstrated.

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INTESTINAL OBSTRUCTION FOLLOWING TECHNICAL ERROR IN PERFORMANCE OF WEBSTER-BALDY OPERATION

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THE operation known as the Webster-Baldy operation for retroversion of the uterus was first introduced by Webster[1] in 1901 and by Baldy[2] in 1903. Although its use has been recommended only in a limited number of selected cases under conditions first emphasized by Polak[3] in 1913, the procedure has been quite extensively and successfully used for the correction of retroversion of the uterus. In 1915 Webster[4] again reviewed his technique for retroversion, intending to emphasize the various stages in the procedure and with special reference to a step which bears repetition in this report. Webster states that in the procedure of the operation, after the round ligaments have been brought through the broad ligaments and have been properly sutured to the posterior surface of the uterus, it is necessary "to close the openings in the broad ligaments, stitching their edges to the round ligaments so that no opening or raw surface is left and there is no chance for the development of a hernia."

In reviewing 100 cases of the Webster-Baldy operations at the Evanston Hospital, not one operative record mentioned the closure of the perforations in the broad ligaments. It occurred to us that an important step was being omitted. Our investigation of the subject was prompted by a recent case which emphasizes the gravity of this error in technique. The case to be presented occurred as a direct but late result of a Webster-Baldy operation. Similar cases have appeared in the literature only twice since the introduction of the operation (Richardson,[5] 1920, and Pemberton and Sager[6], 1929).*

Mrs. O. O., age 45, entered the Evanston Hospital complaining of severe pain in the left lower quadrant of the abdomen, with nausea and vomiting. The onset of her symptoms occurred about six hours prior to admission. She had a normal bowel movement upon entrance and had had two normal bowel movements the previous day. The pain was continuous, not colicky, not relieved by heat, but improved after the use of pantopon. Menstrual history was normal. Further history was negative except that she had had an appendectomy and Webster-Baldy suspension of the uterus fifteen years ago.

Upon physical examination the heart was found to be normal except for occasional extrasystoles; the lungs were normal; the abdomen was not distended, there was a moderate amount of rigidity in the left lower quadrant, with marked tenderness, but no palpable mass. Rectal examination revealed tenderness, especially in the left adnexa. On admission, laboratory findings showed a negative urine and an 11,600 white blood count, hemoglobin 74 per cent, red blood count 3,790,000; temperature

* Since this paper was submitted for publication there has been another case reported by Arnold, L. E. (*Am. J. Surg.* 41: 498–500, 1938) and one of us (J. E. K.) has participated in another case with Dr. J. L. Hagan.

The latter case had a Webster-Baldy operation ten years prior to the date of entrance to the Evanston Hospital and had two full term pregnancies since her operation. She entered the hospital with severe acute pain in her lower abdomen of six hours' duration, a mass in the pelvis, and signs of acute intra-abdominal catastrophe. At operation a herniation of 8 inches of small intestine was found through the left broad ligament. The bowel was viable immediately following release of the obstruction. The postoperative course was uneventful.

99.4; pulse 100; respirations 20, and blood pressure 140/100. Five hours later the white blood count was 15,800, and her symptoms were

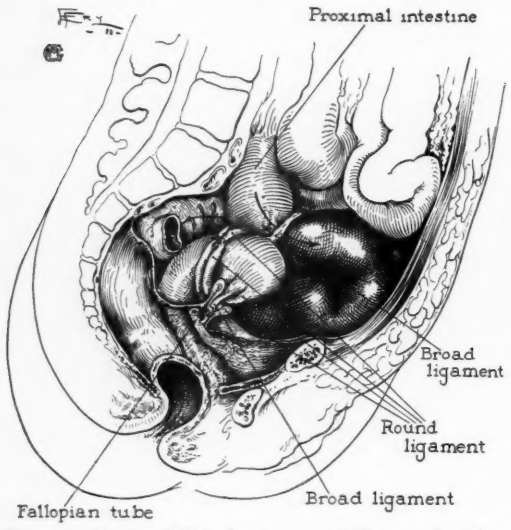

FIG. 1. Case of Pemberton and Sager showing herniation of the small bowel resulting from failure to close opening made in broad ligaments during a Webster-Baldy operation, which is practically identical with the reported case. (From Pemberton and Sager in *S. Clin. North America*, 9: 203, 1929.)

increasing in severity. A preoperative diagnosis of either a twisted ovarian cyst or an intestinal obstruction was made, and abdominal exploration advised.

Through a midline incision there was found in the pelvis a loop of ileum, about 10 inches long, strangulated through the opening in the broad ligament made at the time of an old Webster-Baldy operation. The condition found at operation was practically identical with that pictured by Pemberton and Sager[6] and reproduced herewith. (Fig. 1.) The strangulated portion of the ileum was black and gangrenous, and the peritoneal cavity contained a large amount of dark blood. The obstruction was relieved by removing the left round ligament and Fallopian tube. The strangulated bowel was not viable, and therefore a resection of the necrotic portion was done and an end-to-end anastomosis performed. The patient made an uneventful recovery.

It is obvious that the patient's life was greatly endangered by the technical error in the operation performed fifteen years previously. An explanation of the mechanism involved which led to the herniation of the bowel was suggested by Richardson[5] in 1920. He believed that in his case the broad ligaments may have been too thin, or that the round ligaments were grasped too far from the uterus, or that the perforations in the broad ligaments became too large. Considering these possibilities, it is reasonable to believe that should one of the above conditions exist, a recurrence of the retroversion might result, thus placing tension upon the broad ligaments and tearing them laterally, preparing a potential site for herniation of the bowel. We believe that no Webster-Baldy operation should be performed without following closely the steps emphasized in the original articles of Webster and Baldy. The openings in the broad ligaments should be closed with sutures approximating the cut edges of the broad ligaments and uniting them to the round ligaments. Such a procedure will eliminate any undue tension on the round and broad ligaments and leave no opening to invite herniation.

It has been suggested by Polak[3] that postoperatively one should insert a pessary as a prophylaxis in these cases, its purpose being to keep the cervix back, thus permitting the uterus to assume a more normal anteflexed position, thereby diminishing the probabilities of recurrence of retroversion of the uterus, which probably occurs most frequently in the early convalescent period.

SUMMARY

1. A case of acute intestinal obstruction fifteen years after a Webster-Baldy operation for retroversion of the uterus is reported.

2. The cause of the obstruction was determined to be a failure of closure of the aperture in the broad ligament through which the round ligament was passed.

3. This danger may be prevented by careful observance of the instructions of the originators of the operation.

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5. Richardson, E. P. Intestinal obstruction following Baldy-Webster operation. *Surg., Gynec. Obst.*, 31: 90, 1920.
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The power of the blood to rid itself of bacteria is tremendous, at least partially by virtue of the astonishing phagocytic power of the reticulo-endothelial cells which line the blood channels, particularly of the lungs, liver and spleen. One may inject millions of bacteria or tiny foreign particles into the blood stream, and within a few hours they are found to have largely disappeared.

The brief excerpts in this issue are from "A Textbook of General Surgery" by Warren H. Cole and Robert Elman (D. Appleton-Century).

BICORNUATE UTERUS

DEAD FETUS IN ONE CORNU, NORMAL PREGNANCY IN OTHER

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ON May 29, 1937 a young white woman was referred to me because of a mass in the abdomen, accompanying pregnancy, which was causing severe pain.

The patient knew that she had had a mass in her abdomen for six months, but since her present pregnancy of about three and one-half months, the mass at the side of her uterus had become more painful and seemed to enlarge with the pregnancy. The pain and discomfort was so severe that she had come for advice.

One normal pregnancy had taken place three and one-half years before and a second two and one-half years before. Two years previous she was examined, was told she had a retroversion, and received pessary treatment for a few months. Four months after her last childbirth her menstrual periods became regular and remained so (six day periods) until September 1936, when the period lasted for three days only, and was accompanied by severe cramps. Not long after, the patient became conscious of the mass in the abdomen. She thought she might be pregnant although her menstrual periods continued to be normal and she did not notice the mass getting any larger.

A physician examined her in January 1937 and told her she had a fibroid uterus which should be removed if it became any larger. In March her menstrual period was again only three days in duration, but was not painful. She did not menstruate in April but did in May. She had no further menstrual periods previous to admission to hospital.

Our examination, May 29 and June 23, 1937, revealed a healthy young woman 21 years of age. Her general examination was essentially negative. Abdominal examination revealed that there was a normal pregnancy of about three and one-half or four months' duration. At the right side of the pregnant uterus was a mass which gave the impression of being a fibroid of large size. As the condition was becoming more severe with the progress of the normal pregnancy, we advised a surgical removal under spinal anesthesia.

When the abdomen was opened, July 2, by a midline incision, a uterus with a normal pregnancy of about four months was encountered. On the upper right cornu of the uterus was a mass about 4½ inches in diameter. The right tube with a normal ovary came from the mass, as one would suspect from a bicornuate uterus. But the mass itself was discolored and had the external appearance of a fibroid. Because we did not wish to disturb the normal pregnancy, the entire mass with its accompanying tube and ovary was swiftly though carefully dissected from the pregnant uterus without going deeply into its musculature. The serosa of the uterus was closed over the incision in the uterine wall. The whole procedure including opening and closing the abdomen was limited to thirty minutes.

The patient made an uneventful recovery from the operation, was out of bed in twelve days, and returned to her home in fourteen days.

The mass was examined in the laboratory and the following report made: "Material consists of a large round mass 8 × 11 cm., weighing 220 Gm. It has the gross appearance of a fibroid nodule. On opening this mass there appears a macerated fetus and degenerated placenta two to three months of age. The lining portion of uterus is fairly smooth except at placental site. The wall varies in thickness from ¼ to ½ cm.

"Microscopic sections of the wall of the uterus show some ordinary appearing muscle fibers and give evidence of physiologic hypertrophy of pregnancy. The lining shows hemorrhagic membrane that contains an occasional degenerated chorinic villis.

"Diagnosis: Macerated fetus in cornu of uterus."

On December 15, 1937 this patient was uneventfully delivered with low forceps of a full term, normal infant.

NEW INSTRUMENTS

A NEW TYPE OF FOOT BRACE*

FOR THE TREATMENT OF FOOT IMBALANCES

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THE brace herein described was developed to overcome (1) the disadvantages of the metal supports which are often uncomfortable because of their rigidity, and (2) the shortcomings of the so-called flexible appliances which at best are only palliative, and are entirely devoid of corrective potentialities. This brace may be characterized as a firm support having a slight amount of elasticity. The elasticity is present not only in the insole portion of the brace, a property common to all resilient supports, but is equally present in the inner flange, a property not possessed by other braces.

The basic material of this brace, to which it owes its qualities of firmness plus elasticity, is rawhide. These properties are present in rawhide only in its dry state, for when allowed to soak in water for several hours it becomes sufficiently pliable to permit moulding into any desired shape.

CONSTRUCTION OF BRACE

Thus a rawhide foot brace can be made by moulding the wet hide over a plaster of Paris model of the foot, the negative for which is obtained as for any arch support. The hide is nailed upon the plaster form and allowed to dry thoroughly. It will then maintain its acquired shape as long as it is kept dry. To insure against the moisture of the foot softening and altering its shape, the rawhide brace is treated with celluloid. The brace is then finished by covering it with any of the thin leathers such as kid, calf or suede.

One sixteenth inch thickness of rawhide is able to support the body weight without using any metal reinforcement. An eighth inch thickness of cork, placed under the dome of the insole is sufficient to limit any excesses in the flattening of the arch when the body weight is applied to it. (Fig. 1.) A quarter to an eighth inch wedge of cork, glued to the under surface of the inner side of the heel of the insole maintains the heel in supination. A further aid to the supination of the heel is the inner flange which extends to the back of the heel where it forms a right angle with the insole. This is distinctly different from all other braces (Fig. 1) where the inner flange extends only to the middle of the heel, and thus fails to limit adequately the pronation of the back of the foot. The inner flange in this brace can be extended to the very back of the heel only because of the resiliency of the flange which allows for enough play in the flange to insure tolerability while it hugs the back of the foot snugly.

CORRECTIVE ACTION OF BRACE

Besides giving support to the foot and maintaining the correct foot attitude in a passive manner, this brace has an active corrective force. Its positive action is made possible by the arrangement of the elastic

* From the Department of Orthopedics, New York Post-Graduate Hospital.

insole and elastic inner flange. (Fig. 2.) Were the body weight to act alone upon the brace, it would flatten the insole which would cause the inner flange to move against the foot. Were the pronation of the foot to act alone, it would push the inner flange away from the foot, which in turn would increase the dome of the insole. However, since the body weight and the pronation of the foot act together, and since their forces are exerted at right angles to each other, the result of their simultaneous actions is a force which is located at the junction of the insole and inner flange and is directed upwards against the foot at the astragalocalcaneal level.

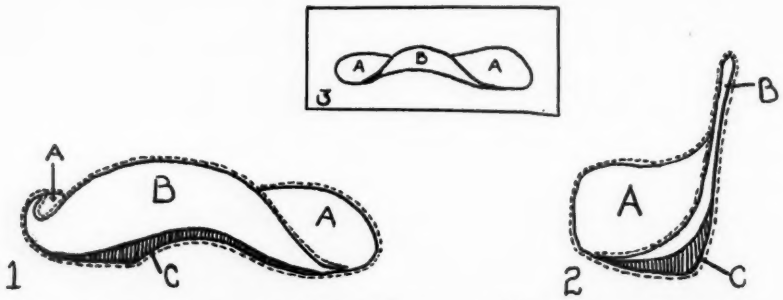

FIG. 1. Construction and design of brace. 1, side view. 2, back view. A, insole. B, inner flange. C, cork wedge. Dotted line represents celluloid coat plus outer leather cover. 3, ordinary arch support. Compare its inadequate inner flange with the inner flange of brace (1) which extends to the back of the foot and partly cups the heel.

This force is not just a theoretic concept but is a real pressure area of which the patient is aware, although it is entirely tolerable. The only way this positive force can be lessened is by having the patient actively assume the correct posture. Whenever the faulty attitude of pronation is resumed, the pressure area becomes evident. Thus there exists in the brace a constant but gentle reminder to keep the foot in the proper attitude.

I have used this brace both in the clinic and in my office for the past six years and have found that it has a wide range of applicability to all types of foot imbalances. It can even be worn with comfort by a patient with spastic fect, a condition which does not tolerate any rigid support. This brace is extremely light in weight, and is comfortable from the very beginning. It does not have to be "broken in" as is so often the case with rigid braces. Corrections can readily be made when necessary by building up the cork wedge beneath the insole. This can be an office procedure, where felt can be substituted for cork.

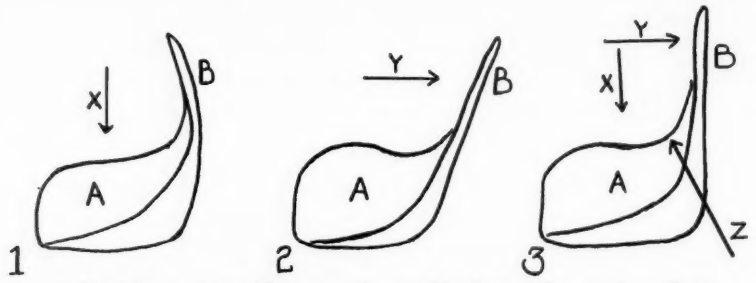

FIG. 2. Positive action of brace. 1, force of body weight, *x*, alone flattens insole, causing inner flange to move in against foot. A, insole. B, inner flange. 2, force of pronation of foot, *y*, alone pushes inner flange away from foot, raising arch of insole. 3, simultaneous action of body weight and pronation of foot produce resultant force, *z*, which is exerted in an upward direction against inner side of foot.

LATHE FOR MAKING BONE DOWELS AND SCREWS

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THIS lathe for making bone pegs and bone dowels provides more accuracy in shaping the dowel and screw, and also saves time.

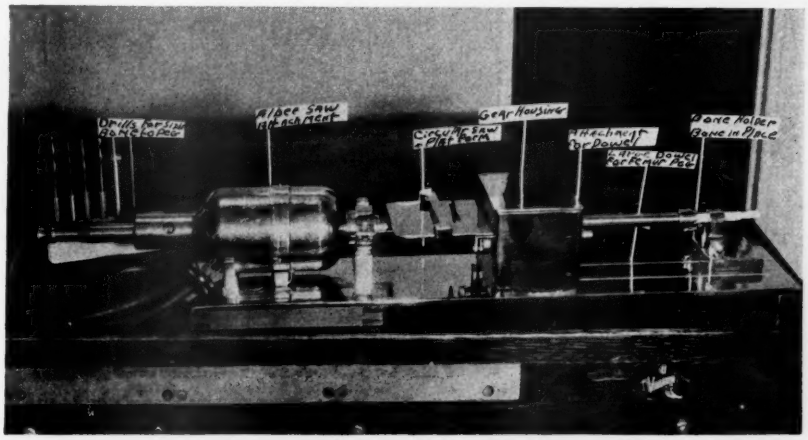

FIG. 1.

My experience with the old type dowel and screw shaper was disappointing in that it consumed too much time and the pegs were not always straight. With the lathe described, time is cut appreciably and there is a regularity in the shape of the peg.

The lathe may be run from any of the motor saw outfits on the market. It may be completely sterilized by boiling, and is finished in chrome-nickel to prevent corrosive action. The gears are placed in a special housing which may be taken down for care after usage. The gears may be oiled with vaseline after each operation. A flange is built under the platform and this flange engages the edge of instrument table so that steady pressure may be made as the bone fragment is fed into the shaper. A small circular saw may be used in shaping fragments of bone before running them through the shaper. This saw has a special attachment and platform for operation.

The bone is fed into the shaper by a small holder which fits into a track on the platform. When fed into the shaper by the holder, the bone is opposite dead center of the dowel. The length of bone that may be fed into dowel-shaper is 3 or 4 inches, so that several pegs may be made at one time. After the dowel is shaped it may be sent through a threader of the size desired. A larger dowel shaper may be used where a peg is to be used for fracture of femoral neck or for intracapsular fractures. The lathe is 22¼ inches in length and 4¼ inches in width.

IMPROVISED APPARATUS FOR UTEROTUBAL INSUFFLATION*

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ATLANTA, GEORGIA

IMPROVISED apparatus for performing the Rubin test are numerous, but most of them lack certain elements of control of pressure and volume of gas to be injected, which is usually air. The following is a description of an apparatus improvised by the author at a nominal cost which uses carbon dioxide from an inexpensive source and places under the immediate control of the operator the pressure and the rate at which it is applied, as well as the volume of gas to be injected. Common laboratory supplies and principles are utilized. No claim for originality is made except in the source of the carbon dioxide.

Two bottles (one-half pint milk bottles are excellent because of resistance to breakage) are stoppered with two-hole rubber stoppers. Through one hole of each stopper a long glass tube passes to the bottom of the bottle and the two are connected by a short length of rubber tubing. Into the other hole of the stopper of bottle A is passed a short glass tube reaching to the lower surface of the rubber only, to which is connected a rubber tube 3 feet in length ending at a stop cock with Luer tip. The remaining hole in the stopper of bottle B contains a Y-tube which reaches only to the lower surface of the stopper. To the arms of the Y are attached 6 inch rubber tubes, one terminating with a female Luer connector, the other with a male connector. The two bottles are bound together with several turns of 1 inch adhesive near their bases. A 5 inch metal rod is passed between the glass tubes in the stoppers and is tightened downward by means of wire loops fastened under the lips of the two bottles. This serves a double purpose of tightening the stoppers and binding the bottles together more securely.

Water containing a dye such as methylene blue is added to bottle A to the upper level of the adhesive band which is marked "0" with a small piece of adhesive. Then 50 c.c. of the water is added at a time and each 50 c.c. level is marked with a small piece of adhesive. A sphygmomanometer with arm cuff detachable from both the manometer and the bulb by Luer connectors is used, the Y-tube of the apparatus being inserted in the place of the bag. A watch for timing the rate of flow is suspended by a cup-hook from the sphygmomanometer frame. A bottle of high grade carbonated water capped with a "fizz cap" used in mixing drinks serves as the source of carbon dioxide for the apparatus. A 2 inch length of No. 14 rubber catheter or pressure tubing of comparable size is attached to the nozzle of the "fizz cap" to receive Luer tip of petcock *a*.

To fill the apparatus with carbon dioxide, petcock *a* is opened and the water in the apparatus is pumped over into bottle A. The petcock is then closed and the control valve of the bulb is opened. The petcock is now connected to the "fizz cap" at a_1 and is again opened. With the bottle upright the "fizz cap" is pressed downward allowing the carbon dioxide to escape into the top of bottle A, displacing the water into bottle B. When sufficient gas is collected in A (the water level should be at the mark "0") the "fizz cap" is allowed to close and petcock *a* is closed and disconnected from a_1. When the blood pressure valve is again closed the apparatus is ready for use. Petcock *a*

* From the Department of Obstetrics and Gynecology, Emory University School of Medicine.

is now ready to be connected to the Rubin cannula and opened.

It will be seen that the rate at which pressure is built up in the apparatus is under the direct visual and finger-tip control of the examiner. The maximum pressure and the volume of the gas to be insufflated is likewise under accurate control. The entire process of insufflation can be instantly stopped with a stroke of the thumb against the control valve of the sphygmomanometer bulb. Once the apparatus is assembled the fittings do not have to be changed and consequently tend to remain air tight. Cost of operation is negligible; we have averaged eighteen to twenty tests from a 28 ounce bottle of sparkling water costing only a few cents. When the carbon dioxide pressure in the bottle gets low, vigorous shaking will aid in driving it out of the solution. The blood pressure instrument can be immediately restored to its original service as soon as each insufflation is completed.

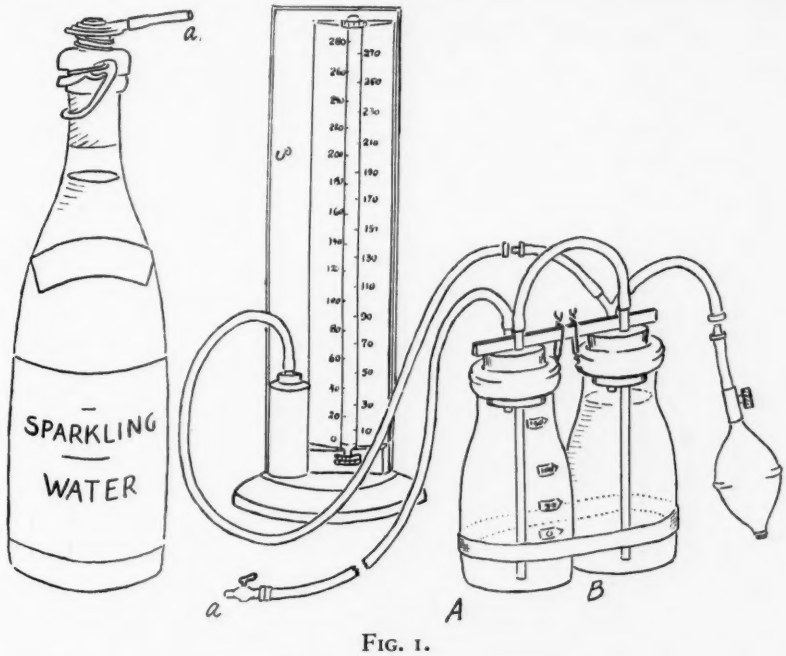

Fig. 1.

A NEW HYPODERMIC NEEDLE SHARPENER

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THE machine here described for sharpening hypodermic needles is driven by a flea power motor (horse power too small to be rated) which consumes electricity equivalent to a 40 watt electric light bulb. It consists of three special emery wheels, coarse, medium and fine. The head of the hypodermic needle is slipped over a small stud and is held in position by a spring clip. The sliding shaft permits the sharpening of a needle of any length or caliber by adjustment of the sliding arm. An adjustable set gauge is attached to the side of the machine which permits one to sharpen several needles of the same length, and give each one of them the same bevel.

A small accessory attachment permits spinal puncture needles with obturators to be sharpened to the proper angle.

A perfectly pointed needle has a manifest advantage over a dull one. A sharp needle is particularly needed for extensive intradermal work since the dull needle produces more reaction and more trauma to the tissues. Intravenous work also requires well pointed needles, especially where there is considerable adipose tissue and the veins are small.

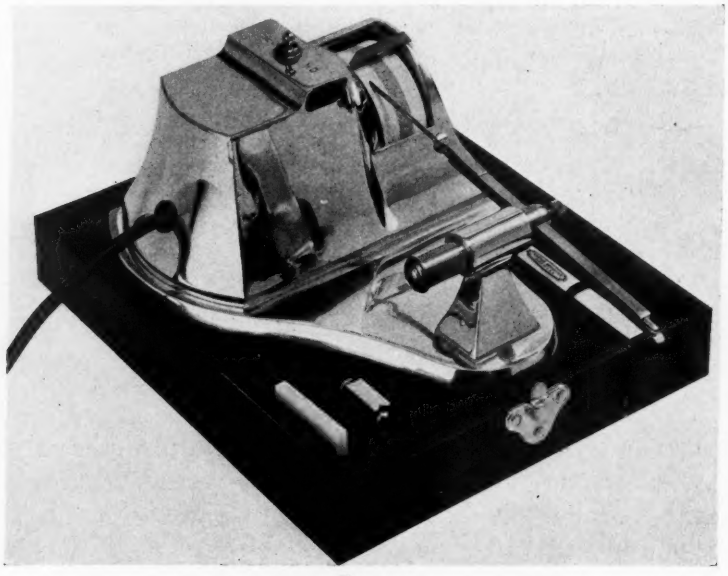

FIG. 1.

The present machine will give a hollow ground point, with any degree of bevel desired, to any hypodermic needle regardless of length or caliber. The slight burr which is caused by the first grinding of the needle is easily removed by a few light brushes across the furnished Arkansas stone.

Several sizes of clean out wires for partially obstructed needles are furnished. These are of spring steel and will not kink.

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

INCUNABULA MEDICA II—THE YEARS 1001–1100 A.D.]

MONTE CASSINO AND THE SCHOOL OF SALERNO

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IT is to western civilization that one must turn to know and understand Constantinus, the man of learning from another continent. The beginning of the mass folk migrations had already taken place. Wave following wave, Celtic and Teutonic tribes had moved southward and westward, overrunning the outworn Roman empire, and like the Mongols and the Turks in the East, adopting the civilizations and the creeds of those whom they conquered. It was Christianity which gave these new developments their stamp. The glad tidings of a Christ had come to the sick as well as to the hale and hearty. Illness was freed from the odium of sin and the inferiority which had clung to it from primitive times and throughout the days of classical culture. Suffering was a mark of God's own grace and denoted a degree of transfiguration. The sick patient became a privileged person. Through compassion, the healthy could participate in his privileges. Indeed, it became a Christian duty to do all that could be done for the poor and the sick.

In due time the care of the sick was organized upon a grand scale. Great hospitals were built. Benedict, founder of the great order named after him, instructed the monks of his order to pay special attention to the care of the sick.

Although at the outset, Christianity was fundamentally hostile to classical science, by degrees the new faith became reconciled to it, and reconciled even to pagan medicine. Obviously, a Christian equipped with medical knowledge could be more helpful to the sick than could an ignoramus. Cassiodorus, who held high office under Theodoric, and who subsequently became a monk, declared that a study of classical literature was meritorious for a Christian. The library of the monastery in which he lived to a great age, contained among a vast collection of manuscripts, many of medical content. The Benedictins gradually adopted Cassiodorus' principles, with the result that wherever Benedictin monasteries were established, collections of all available medical and philosophical writings were made, and these monasteries became centers for the study and practice of medicine, places to which the sick were welcomed for Christ's sake, and where likewise the medical lore of the ancient world was studied, insofar as it had been preserved from destruction. Among such

centers, St. Gall and Reichenau were worthy of special mention.

In the early Middle Ages, doctors were

Fig. 1. Title page from Rhazes "Liber Nonus ad Almansorem" printed in Padua by Peter Maufer in 1480. At Monte Cassino and Salerno the texts of Rhazes enjoyed their greatest popularity.

mainly members of various holy orders. Doubtless there were lay practitioners as well, but they formed an infinitesimal minority. Priests and monks were mainly responsible for keeping the literature of medicine alive, as well as for being medical practitioners. Therefore, there is good reason for speaking of this period as that of "monastic medicine."

In the East the works of the great Greek physicians had been very early translated into Arabic, and their contents assimilated. They had long remained closed books, however, to the Latin speaking West. No doubt some of the writings of Hippocrates, Galen, and other Greek doctors had been Latinized, but they were much too complicated, and had secured therefore but little acceptance. Much more successful in distribution were brief treatises, extracts, and condensations from the Greek masters, making certain essential facts immediately available for the practice of medicine. The upshot of all this was that the early medieval literature of medicine consisted mainly of collections of recipes, of brief dietetic hints, and of concise monographs upon the pulse, fever, the urine, and bloodletting. These were sometimes couched as letters, sometimes in catechismal form, and sometimes penned in verse, thereby to be remembered the more easily.

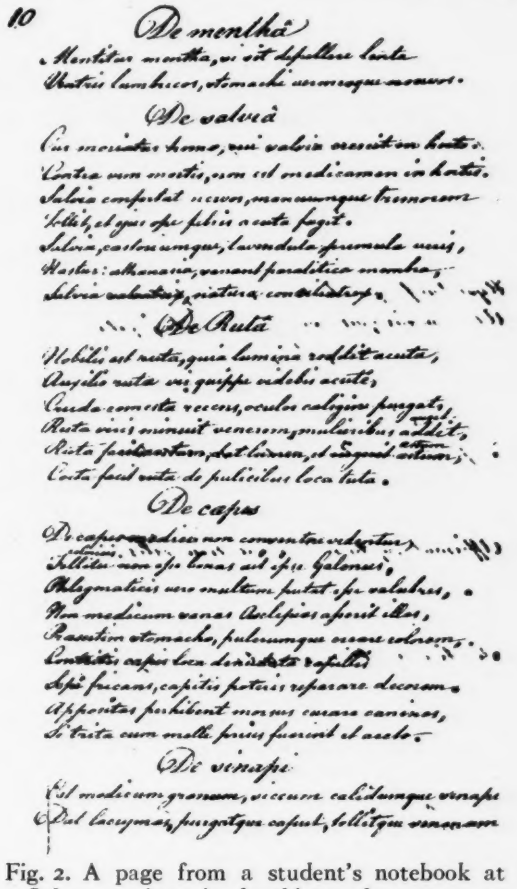
We shall look in vain in the medical literature of those days for original ideas. They had been lost in the process of transition, but the medical teachings already extant and those in actual practice daily, were scrupulously preserved. Nor were these blindly transcribed. The monastic medical authors made compilations, indeed, but did so understandingly, and incorporated their own knowledge of popular medicine into the text. The great incalculable service of the Church to posterity at this time was that it not only undertook the care of the sick, and organized it, but even more that in the Dark Ages of misunderstanding, it kept the spark of medical science glowing so that, when the time became ripe, new fires could be kindled. It was not, however, until the second half of the Middle Ages had begun, that there came a man, an African named Constantin, therefore called Constantinus Africanus, who was to give a decisive impetus to medieval medicine.

One cannot but hesitate to describe Constantin as one of the "great doctors." History really knows nothing whatever about his capacity as a physician. His importance to medical science did not lie in the field of actual practice, though nonetheless his medical works and writings had great influence. Who was this Constantin, arriving on the scene from another continent with new ideas, new concepts, strange contacts?

He was born at Carthage, somewhere about the year 1010. Legend clings to his

personality as was customary in those times. His biographer, Petrus Diaconnus, informs the world that he visited Babylon, to study there the medical and other sciences of the Chaldeans, the Arabs, the Persians, and the Saracens; that from Babylon he went farther afield into India, whence he returned by way of Ethiopia and Egypt, absorbing on his travels the wisdom and lore of these lands. It is written that his journeyings lasted for thirty-nine years, and that he was heavily burdened with knowledge when he returned to his native city. His compatriots, however, regarded him as a sinister figure, and they planned to put him to death. Having got wind of such an intended assassination, he took refuge on board a ship sailing to Salerno. He remained there for a time in hiding, disguised as a beggar. Then it so happened that the brother of the King of Babylon passed through Salerno, and recognized Constantin, in spite of the disguise, and he was then treated with great honor at the court of Robert Guiscard. He did not find court life congenial or to his liking. Having heard glowing reports of the intellectual life at Monte Cassino, he determined to journey there. On his arrival he secured a very friendly reception from the Abbot Desiderius, so that he decided to prepare himself to become a monk. In the peace and tranquility of the cloister, he devoted himself to the translation of medical works from Arabic into Latin, until, in the year 1087, at his death the pen literally dropped from his hand. His fellow monks chose to remember him as "magister orientis et occidentis," as a new Hippocrates.

This is about all we know about Constantin of Africa; not much, and yet it is sufficient for the present. The account is, of course, legendary, but, like all legends, it contains a kernel of truth. A truer account will be given in another chapter. There can be no question but that Constantin came into close contact with the world of Islam, that he studied Oriental science, and transmitted it to the West. There exists also ample evidence that he was as well informed in the western science of his day, and it is known that he wrote in excellent Latin, differing greatly from the barbaric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De menthâ
De salviâ
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De sinapi

Fig. 2. A page from a student's notebook at Salerno, written in the thirteenth century.

phrasing of the monastic medical writers of previous centuries.

Constantin's medical significance lies in his role of intermediary. As if with a magic key, he opened the world of the East to the West. Medical literature had become dry and arid. The monastic medical writers had been chewing over the same texts for half a millennium. Although these texts had sufficed for a time to guide medical practice, they were being superseded. Medicine was engaged in the act of breaking away from the Church. Education was no longer exclusively a clerical affair; there existed now educated laymen as well, and they had nothing to hide, so brought much into the light. Centuries were still to elapse before independent productive work was once more to become possible, but intelli-

gent men were thirsting for better instruction. Constantin had made it accessible to them. Medical men were enabled, by his

De Anima Folio 1.

Fig. 3. Title page of Aristotle's "De Anima" printed in Cologne in 1490, by one Quentell. In manuscript form prior to the invention of printing, it comprised one of the most popular medical texts at Salerno.

translations, to read the writings of the great Arabic physicians, though not those of Rhazes, or Avicenna, which would have been beyond their comprehension. The medical men were still in the kindergarten stage. The works translated by Constantin were those of much less noted authors, tenth century writers such as Isaac the Jew, Ali Ibn el-Abbas, etc.

By the same Arabic route, the practitioners of his day became acquainted with the writings of the old Greek physicians, with the aphorisms of Hippocrates, his Prognosis, his Dietetics—books which many hundred years before had been translated into Latin, but had not been understood and had therefore been forgotten. Returning anew to the West by way of the East, they were eagerly read. With Hippocrates came Galen, Galen's commentary on the Hippocratic writings, his great work on therapeutics, and his little Ars Medicus. Galen, who already dominated Oriental medicine, began now to achieve the conquest of the healing art in the western world.

What gave Constantin's translations so speedy an influence was that they became textbooks at the School of Salerno, where they appeared at an apt moment. Salerno was a flourishing seaport lying not far to the south of Naples. It was an archepiscopal see, had a famous cathedral, and many monasteries. In these times it had become widely known for its medical school. As early as the beginning of the tenth century, physicians of Salerno had acquired a fair measure of reputation beyond the boundaries of their principality, not merely as men of learning, but as competent practitioners of medicine. Diversified influences were at work in the place. Classical learning had never completely died out there, although it was dying. Greek was still understood, forming a supplementary language to the local Italian dialect and the Latin of the learned. There was a brisk trade between the town itself and the Orient. Arabic knowledge found its way into the place mainly by way of the adjoining Island of Sicily. There is a legend relating to the foundation of the School, to the effect that one day four doctors, a Greek, a Roman, a Saracen, and a Jew, decided upon the joint composition of a book of recipes or prescriptions, and thereby founded the school. This story gives a key to the characteristics of Salerno. It was a place where laymen and clerics of varied religions and nationalities, therefore opinions, could collaborate in friendly rivalry.

In this little community of Salernian physicians, the translations of Constantin had become known. The fertilizing effect was amazing, the growth astounding. Those who had been no more than ordinary family practitioners became suddenly men of great learning. Salerno developed into a "civitas hippocratica," just as, at an earlier date, Constantinople had in the

East. A medical faculty, the first medical faculty of the West, came into being. During the twelfth century the School gave birth to an extensive literature; prescription books, textbooks of special pathology and therapeutics, monographs upon fever, the urine, the pulse, diet, treatises on surgery and on gynecology. Anatomy was once more studied, after having fallen for some time into disrepute. Animals, especially swine, were prepared for anatomic dissections and demonstrations, and brief handbooks on the subject were penned. Throughout these writings, in every line, was felt the influence of Constantin. The new knowledge which he had brought to the West was being elaborated upon, the teachings of the Greeks and of the Arabs were being harmonized with the experience of the medical men of Salerno. But the latter felt humble-minded in the face of the achievements of the great masters. How could they have done otherwise? Even today, we of the twentieth century should do likewise.

We should err were we to suppose that the Salernians of the twelfth century did original scientific work, or that it would have been reasonable to expect such work from them. Their main object was to learn. They learned the art of the practice of medicine at the bedside. They learned their facts and prescriptions from the new literature made available to them. Working over and over again on what they had learned, they passed it on to others, sometimes by word of mouth to the pupils, who came in ever increasing numbers to Salerno, sometimes in their books and writings, in the new medical literature they produced. This new literature was enormously superior to the writings of earlier monastic medicine, therefore easily supplanted it. They saw no reason for formulating new theories. The Greco-Arabic science, whose wealth had so recently and surprisingly been disclosed to them, provided sufficient explanation of all things, and fully gratified their scientific thirst.

The names of many of the Salernian physicians became so well known over the world, and were so indelibly imprinted into medical achievement, that even today they are fairly familiar. Such were Fra Bartholomaeus, better known as Bartholomeus the Englishman, about whom we shall write in a later chapter, Thomas Aquinas, the Angelic doctor, Platarius, and Copho, also Magister Ferrarius. It is difficult to discern the actual human beings behind these great names. Little can be learned concerning their individual lives, and their books and manuscripts seldom convey a personal note. The work of the School of Salerno resembles that done by a guild of artisans. That is why no attempt has been made here to cast any one of the Salernians in the rôle of the great doctors. We merely make brief reference to the School as a whole, an account of single individuals being supplementary entirely to a chapter upon Constantin of Africa.

Moreover, the School of Salerno had but a very brief but blossoming rôle, that of an instructive, let us even say a stimulating impetus in the history of medicine. It flourished most abundantly during and shortly after the days from 1050 A.D. to about 1200 A.D. In 1195, the town of Salerno was sacked by the Emperor Henry VI, and the school suffered therefrom. Thirty years later, in 1225, the University of Naples, which was to become a formidable rival, was founded by the Emperor Frederick II. Even after this, however, Salerno managed to maintain its reputation for a time, to such an extent that, according to ordinances issued by Roger II of Sicily and by Emperor Frederick II, no doctors were to be allowed to practice within their particular realms unless they had received the "approbatur" of the physicians at Salerno.

BOOK REVIEWS

DIAGNOSTICS BIOLOGIQUES. By Noël Fiessinger, M.D., Henri-René Olivier, M.D., and Maurice Herbain. Fourth Edition. Paris, 1938. Maloine.

Professor Fiessinger's "Laboratory Diagnosis" has run through four editions and gained two French scientific awards—and has earned both. It is a laboratory manual with a difference. It is aimed to provide for the needs of the physician far away from large medical centers, and shows him how, with a minimum of equipment and cost, he can perform the diagnostic work necessary to give complete care to his patients.

In his introduction to the first edition, the author set himself the task of presenting for each procedure: (1) The materials necessary (*Ce qu'il faut*); (2) The procedure (*Ce qu'il faut faire*); (3) What to look for (*Ce qu'il faut voir*); (4) How to interpret (*Ce qu'il faut dire*). To this end each test is taken up under the four headings and the points are presented with Gallic clarity and powers of organization.

This is one of the few manuals of this type which brings into one volume the procedures of bacteriology, mycology, parasitology, vaccines, hematology, and tests relating to gastro-intestinal, respiratory, cardiovascular, metabolic, renal and nervous function.

It is probably doubtful whether an average practitioner could find the time or the occasion to perform all these tests. However, a book of this type prepares him to handle intelligently the work he must do and to interpret intelligently the work done by others. It is literally "multum in parvo."

SURGICAL TECHNIQUE AND PRINCIPLES OF OPERATIVE SURGERY. By A. V. Partipilo, M.D., F.A.C.S. Third Edition. Chicago, 1938. Chicago Post-Graduate School of Surgery. Price $10.00.

Ten contributors discuss different aspects of surgery in this third edition of Dr. Partipilo's book on Surgical Technique, the first appearance of which was in 1930. It is a serviceable book for the intern on a surgical division, for the resident in surgery who wishes a reference book for points of technique, and for the general surgeon-physician who finds a certain amount of surgery necessary in his practice.

There are thirty-three chapters (567 pages in all) covering every phase of the subject. The 404 illustrations by W. C. Shepard are excellent. Parts of the work are revisions and parts are entirely rewritten. It is therefore timely and up-to-date.

SURGICAL ANATOMY. By C. Latimer Callander, M.D. Second Edition. Philadelphia, 1939. W. B. Saunders. Price $10.00.

This is an excellent book, now presented in a second and entirely reset edition. The first edition won universal praise from book reviewers and this praise was echoed by those who bought the volume. What more can or need be said?

Anatomy and its surgical application are considered together or in close sequence. The writing is uniformly good. The illustrations are excellent and in every case serve a purpose. The book is 858 pages long and has an ample index.

To both student and surgeon it represents a book which definitely belongs on the library shelf, to be referred to many times in the course of the day's work.

ALCOHOL IN MODERATION AND EXCESS. By J. A. Waddell, M.D. and H. B. Haag, M.D. Richmond, 1938. William Byrd Press. Price $1.00.

In response to a resolution passed by the Senate and House of Delegates of the Legislature of Virginia in 1936 that called for "accurate information as to the effect of the use of alcohol upon the human system, in respect to both moderate and excessive use thereof," researches were made which are recorded in this book.

The chapters cover, "Introduction to the Study of Alcohol," "The Action of Alcohol on the Body," "The Action of Alcohol on the Central Nervous System," "The Action of Alcohol on the Other Body Systems," "Miscellaneous Topics Pertinent to the Action of Alcohol on the Body," "Acute Alcoholism and

Chronic Alcoholism," and "Statistical Data Pertinent to Alcohol and Its Effects."

This work (184 pages, indexed) compiled by eight distinguished Virginia medical educators is an unbiased and valuable presentation of the subject.

Chronic Diseases of the Abdomen. By C. Jennings Marshall, M.D. New York, 1939. Little Brown.

Donald C. Balfour in the Foreword says, " . . . it is immediately apparent that Dr. Marshall has succeeded admirably in what he set out to do, namely, to make available to the practitioner a small but comprehensive volume in which significance of signs and symptoms as they are related to abdominal diseases can be readily found."

This book consists of 247 indexed pages, of which Part I is concerned with case taking and physical examination, Part II with differential diagnosis.

It is not simple to diagnose some of the chronic diseases of the abdomen, and this well written book, based on long clinical experience, contains valuable aids in this direction. The majority of surgeons and internists could profit by it and—it is not only instructive, but it is good reading.

A Treatise on the Surgical Technique of Otorhinolaryngology. By Georges Portmann, M.D. Translated by Pierre Violé. Baltimore, 1939. Williams & Wilkins. Price $12.50.

This is a splendid work for any physician who does ear, nose and throat surgery. It is a translation of Portmann's famous work published in France, and, we remark in passing, the translator has done a creditable piece of work. The book has been revised and adapted to American conceptions, we are told, and new procedures have been added. Not knowing the original, we have no means of judging. However, the technique of surgical procedures in this field are described; the writing is clear, fast moving, stripped of padding, and comprehensible.

The book is 675 pages long, beautifully printed (we are not quite used to double columns in books, but it makes for easy and speedy reading), has 474 illustrations and two colored plates. Bibliography and index are, unfortunately, lacking.

Emotions and Bodily Changes. By H. Flanders Dunbar, M.D. New York, 1938. Columbia University Press. Price $5.00.

This work first appeared in 1935 and presents a survey of the literature on psychosomatic interrelationships from 1910 to 1933. In this, the second edition, there is a supplementary introduction and additional bibliography.

This bibliography brings together in some sort of perspective the research material that has combined to develop those concepts in medicine which are the necessary foundation for further research in the domain of psychosomatic problems. Part 1 deals with orientation and methodology; Part 2 summarizes the literature relating to emotions and organs or organ systems; Part 3 contains therapeutic considerations and concluding remarks.

This work should prove of inestimable value to those whose field is the psychological aspect of disease. The author reveals great insight and there is evidence throughout the work of extensive research into the relevant literature. The influence of this book is certain to be wide and telling in the field of mental medicine.

Surgical Anatomy of the Head and Neck. By John Finch Barnhill, M.D. Baltimore, 1937. William Wood & Company.

This large book (921 pages) will be of interest to the student of anatomy and of great value to the otolaryngologist, the oral surgeon, the dentist and the ophthalmologist. Internists and those vaguely labeled as "diagnosticians" may well refer to it with profit.

The volume itself covers the subject in detail in an interesting and inspirational manner. Although a textbook, it lacks certain elements common to most textbooks. It is not dull and flat, but is stimulating. It is good reading. The style is different; we would have more books written in the author's style and manner. The type is large. The illustrations (431) are good, the colored ones very good. There is an ample index.

This is a *surgical* anatomy, an anatomy that has a practical value to the oral or head

surgeon, the man doing otolaryngology, and to the surgeon-dentist.

THORACIC SURGERY. A revised and abridged edition of Sauerbruch's Die Chirurgie Der Brustorgane. By Ferdinand Sauerbruch, M.D. and Laurence O'Shaughnessy, M.D. Baltimore, 1937. William Wood & Company. Price $13.50.

The author of this important book needs no introduction to American surgeons who are well aware of the brilliant pioneer work of Professor Sauerbruch and of his influence on the development of thoracic surgery. In 1904, due to the introduction of differential pressure anesthesia, Sauerbruch and those associated with him occupied themselves with the problems of chest disease. The results of these experiences were reviewed in the first comprehensive work on thoracic survery (Die Chirurgie der Brustorgane), which came out in no less than five editions (1918 to 1930). The present volume is a revised and abridged edition of the larger parent work.

The book is 394 pages long, the illustrations (some in color) are excellent. The translation is scholarly and the abridgment reveals good editorial judgment. There is an extensive bibliography and a subject index.

Any surgeon who is concerned with thoracic surgery should own this work and look upon it as his thoracic surgery Bible.

GROSS ANATOMY. A Brief Systematic Presentation of the Macroscopic Structure of the Human Body. By A. Brazier Howell, M.D. New York, 1939. D. Appleton-Century Company. Price $6.00.

Throughout a considerable period of years the hours allotted to the course in gross anatomy in American medical schools have been progressively curtailed. Yet it is impossible to teach the student "all about gross anatomy." One cannot cram the subject. Once upon a time, when a thousand hours (not the present three or four hundred) were allotted to this subject the student bought and studied a textbook. Today, such works are not textbooks in the true meaning of the term, but reference works. It is impossible for the student to differentiate the important from the unimportant. He learns the book rather than anatomy. Often he relies on a dissecting manual—a practice as pernicious as the employment of a "trot" to the student translating Latin.

With these faults in mind the author has boiled his subject down to the bare essentials and has stressed the underlying principles, clarifying the points which usually puzzle the majority of students. The book is concerned with gross anatomy only, omits histology entirely, and includes only that amount of neurology needed for a proper understanding of the gross field. The author tells us that "It is an exposition of the procedure followed in the course on that subject as given during the 320 hours of instruction in the Medical School, Johns Hopkins University."

We hope students in other medical schools will have this work included in the books they "must" purchase. This book has a service to perform to those who find anatomy a dry, dull subject, a feat of pure memory.

SPECIAL SYMPOSIUM

SURGERY OF THE BILIARY TRACT

BY

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SURGERY OF THE BILIARY TRACT*

A STUDY OF THE FACTORS IN SURGICAL MORBIDITY AND MORTALITY

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SINCE the first successful "operation for gallstones," reported by Dr. Bobbs in the June 15, 1867 issue of the *Indiana State Journal of Medical Science* and the first successful cholecystectomy by Langenbuch[1] in 1882, the progress made in the treatment of the surgical diseases of the biliary tract has been phenomenal.

This study involved the critical examination of 5,200 consecutive cases discharged from the Post-Graduate Hospital with a diagnosis of cholecystitis in any form and of any other pathology of the gall-bladder or the bile ducts. Of this group 3,986 were treated surgically and these were examined in detail with especial reference to: (1) past history; (2) present history; (3) physical and laboratory findings; (4) type of surgery; (5) clinical and microscopic pathology; (6) bacteriology; (7) course in the hospital with complications; and (8) mortality. In this paper only the most obvious factors influencing the morbidity and the mortality in this series of cases will be discussed. More emphasis has been placed on acute cholecystitis for it is felt that this represents the gravest problem among the more common diseases of the gall-bladder.

Table 1 is a summation in terms of operations and their respective mortalities of all the patients operated on in the Post-Graduate Hospital from January, 1920 through June, 1937. This classification of operations is entirely on the operative procedure and the pathologic determinations were made independently by the attending pathologists. This group included every operation performed on the biliary tract irrespective of preoperative complications and secondary or even primary conditions, the economic status, age and debility of the patient, and regardless of whether the operation was elective or imperative. The distribution between private and clinic patients was nearly 50-50 and while the obvious difference in mortality and morbidity in the two groups was manifest here, it is believed that this factor should have no bearing on the interpretation of the results. It is further our belief that no mere comparison of statistics has any value unless all the factors above mentioned are taken into consideration.

Heuer,[2] in a comprehensive statistical review, collected 36,623 biliary tract operations in which the mortality was 6.6 per cent. Judd and Parker[3] as early as 1924 published a report of a consecutive series of operations on the gall-bladder with a mortality of 1.8 per cent. Less than 4 per cent, however, were acute infections. From that low mortality in a general series, mortality rates are quoted up to 14.2 per cent in one metropolitan hospital. It is obvious that these authors are speaking of dissimilar conditions, with varying severity of disease, stage of surgery, etc.

The gross mortality for biliary tract surgery in our study was 7.7 per cent. In Table 1 the surgery is divided into three groups, i.e., (1) surgery for the chronic gall-bladder, including nine operations for carcinoma of the gall-bladder; (2) surgery for malignant (permanent) obstructions of the bile ducts, primarily the common duct; and (3) surgery for acute cholecystitis.

* From the Gall-Bladder Clinic, Departments of Surgery and Medicine, New York Post-Graduate Hospital (Columbia University).

Cholecystectomy, with or without appendectomy, for the chronic gall-bladder, accounted for 2,438 operations, by far the largest part of the total number. The mortality in this group was 3.61 per cent, which is definitely higher than in many recently published series, including those personally observed by Heuer[4] and particularly those from the Mayo Clinic.[3]

TABLE I
ANALYSIS OF 3,986 CONSECUTIVE OPERATIONS ON THE BILIARY TRACT (1920–1937)

Operation	Total Number	Mortality (No.)	Mortality (Per Cent)
Chronic Cholecystitis			
Cholecystectomy	3,240	190	5.8
Alone or with appendectomy	2,438	88	3.61
With dochostomy	238	27	11.34
With other operation	568	74	13.03
Cholecystectomy with dochostomy and other operations	6	1	16.16
Cholecystostomy	66	22	33.3
Alone or with appendectomy	43	13	30.24
With dochostomy	16	6	37.50
With other operation	7	3	42.80
Total	3,306	212	6.40
Obstructive Biliary Disease			
Cholecystogastrostomy	52	15	28.8
Choledochostomy only	37	13	35.1
Choledochostomy with other operations	2	2	100.0
Plastic on ducts	5	3	60.0
Total obstructive	96	33	34.4
Acute Cholecystitis			
Cholecystectomy	517	45	8.82
Alone or with appendectomy	428	32	7.47
With dochostomy	89	13	14.60
Cholecystostomy	45	13	28.80
With dochostomy	9	3	33.33
Total cholecystostomy (with 3 other operations)	57	18	29.63
Total acute cholecystitis	574	64	10.97
Total for all biliary tract operations	3,986	309	7.7

Cholecystectomy with dochostomy was resorted to in nearly 7 per cent of all gall-bladder excisions. The gross mortality was 11.34 per cent. This represents apparently a conservative estimate of the risk of invading the common duct. Smith[5] gives a mortality of 33 per cent for common duct stone with drainage, in ninety-eight common duct cases including twenty-two with acute gall-bladder. Glenn[6] reports forty-two cases with a mortality of 9 per cent. The noteworthy feature, aside from the mortality, is the frequency of exploration of the common duct. In 86 per cent of these cases common duct stones were removed.

In all but eight of the 238 cases drainage was employed, the method of drainage varying with the surgeon. The index of 7 per cent common duct intervention represents either the general severity of the cases operated upon in this series or the thoroughness of the surgery. Maingot,[7] in a recent plea for more frequent exploration of the common duct, states that the mortality of cholecystectomy should not be materially increased by so doing if no pathology is found. He places the probable mortality at 2 per cent. In this group the common duct stone cases had a mortality of 12.8 per cent and those without stones 3.8 per cent or not materially greater than for cholecystectomy alone.

The problem of multiple surgery arises in all operative series, but is all too rarely discussed. In 568 operations cholecystectomy was combined with one or more other operative procedures. Only such operations as within themselves carry a possible mortality were included. These "secondary" operations range from resections of the stomach to hemorrhoidectomy. Purely minor secondary surgery, such as the removal of a fibroma or a mole, are not included. The astonishingly disproportionate mortality of 13.03 per cent in this group deserves further consideration.

The infrequent use of cholecystostomy is significant. Only sixty-six operations of this type were performed, with a mortality of 33.3 per cent. They represent but 2 per cent of the operations on the chronic gall-bladder. Macroscopically, from the clinical pathologic standpoint, the gall-bladders on which cholecystostomy was done were as a

rule no more pathologically advanced than those enduring cholecystectomy, but the operative risk was higher. Therefore no comparison is intended. In spite of the attempt of some continental surgeons, notably Juraristi,[8] to popularize cholecystostomy, it has been infrequently resorted to. That the trend continues toward cholecystectomy can be well illustrated by the extensive study by Mentzer,[9] who reported 55.7 per cent cholecystostomies in 1906 and only 4.3 per cent in 1926. Eliason and Ferguson[10] have shown the futility of the operation, except as an emergency measure.

OPERATIONS FOR OBSTRUCTIVE BILIARY TRACT DISEASE

Operations primarily for obstruction in the duct system numbered ninety-six, with a gross mortality of 34.4 per cent. Fifty-two anastomoses circumventing the lower common duct were performed. Of these, fifty were cholecystogastrostomies and two were cholecystoduodenostomies. The mortality rate was 28.8 per cent. Thirty-six (71 per cent) were for carcinoma of the pancreas, and the remainder for obstructive pancreatitis. All patients were severely jaundiced. This mortality is singularly like that reported by Bernhard,[11] and by Collier and Winfield.[12] Judd and Parker,[13] and Eliason and Johnson,[14] had slightly higher mortality rates using a number of procedures (38.2 per cent and 40 per cent respectively).

Choledochostomy following a previous cholecystectomy was done in thirty-nine instances, with a mortality rate of nearly 40 per cent. This further emphasizes the contention of Maingot[7] for exploration of the common duct in all cases of cholelithiasis in order to avoid the high mortality of a subsequent operation on the common duct. Only six of the thirty-nine patients had previously been operated on at the Post-Graduate Hospital. Thirty-two of the group had recurrent or overlooked stones. The remainder had stenosis of the common duct.

CHRONIC CHOLECYSTITIS

Table II is a summary of the primary factors influencing the morbidity and mortality in the operations for chronic cholecystitis presented in this study. For purposes of comparison, a "morbidity factor" has been devised as an index of the immediate postoperative complications. A factor of 1 was given to cases with relatively uneventful recovery, 2 to those with complications usually not influencing the mortality (wound infections, bronchitis, abnormal distention, etc.); and 3 to those with complications severe enough to cause death in themselves (pneumonia, dehiscence, atelectasis, acute cardiac failure, etc.). The general morbidity factor for the entire series of operations on the chronic

Table II

CHRONIC CHOLECYSTITIS

Factors Influencing the Morbidity and Mortality in Surgery for Chronic Cholecystitis

	No. of Cases	Morbidity	Per Cent Mortality
(A) Multiple surgery (Cholecystectomy plus secondary operation)	575	1.72	13.85
1. With gastroenterostomy	128	....	16.4
2. With pyloroplasty	111	....	9.9
3. With gastric resection	61	....	31.1
4. With acute appendix	31	....	13.0
5. With hysterectomy	59	....	11.8
(B) Conservative treatment in acute cholecystitis			
1. Subsided acute	474	1.59	11.02
2. With chronic abscess or old perforation into colon	46	2.20	38.0
3. Cholecystostomy in previous acute	15	1.58	20.0
(C) Cholecystostomy in chronic*	68	1.50	7.4
(D) Jaundice (especially necessitating common duct surgery)	254	....	13.0
(E) Delay in surgery for chronic cholecystic symptoms. Series with less than two years' history	959	....	1.35
Total series of chronic cholecystitis	3,303	1.31	6.4

* Means cholecystostomy for a previous chronic infection—now followed by a cholecystectomy.

gall-bladder was 1.31 and the general mortality was 6.4 per cent.

Foremost among the factors influencing this morbidity and mortality was multiple surgery. The morbidity in this group of 575 cases was 1.72, or 135 per cent higher than in the general group and nearly 300 per cent higher than in those cases in which cholecystectomy alone was done. The mortality rate of 13.85 per cent was nearly four times as high as that for cholecystectomy alone. In only eight of these cases was the gall-bladder pathology of such severity that multiple surgery was imperative. These were patients in whom the gall-bladder had perforated into a segment of the gastrointestinal tract with obstruction. Uniformly the gall-bladders removed in instances of multiple surgery had a relatively severe degree of pathology.

Three hundred of the 575 multiple operations were for concomitant disease in the gastroduodenal segment. Only twenty-four were for associated malignancy of the stomach. Thus 9 per cent of the operations done for chronic cholecystitis had surgery for associated gastroduodenal pathology, mainly ulcer. How adversely the addition of cholecystectomy to operations on the stomach affects the mortality can readily be seen in Table 11. Cholecystectomy plus pyloroplasty had a mortality of 9.9 per cent. Cholecystectomy plus gastroenterostomy had the disproportionately high mortality of 16.4 per cent. With gastric resection added to a cholecystectomy the mortality rate was 31.1 per cent. Niemeier[15] reported but 2 per cent ulcers in a series of chronic cholecystic cases, while others have reported an incidence varying from 0.9 to 10 per cent. Heyd,[16] in a personal series reported from this hospital, gives the incidence of ulcer as 10.5 per cent in 557 operated cases. The association of chronic cholecystitis with ulcer of the stomach and duodenum is receiving increasing attention in the current literature. That this association in surgery carries a prohibitive mortality in a large series of cases is therefore of great importance.

Thirty-one patients had their gall-bladders removed in the presence of a pathologically acute appendix and in four of these the appendix was perforated. The mortality rate was 13 per cent. In no instance was the gall bladder acute, though in the majority of instances it was of a severe pathology. The mortality rate of 11.8 per cent where hysterectomy and cholecystectomy were done is further evidence that the patient with cholecystitis cannot well tolerate additional surgery.

There were 474 cases with definite pathology indicative of a previous acute cholecystitis. All had subsided completely before operation. In spite of this, the mortality rate was 11.02 per cent. It is interesting that 437 of these patients gave a history of one or more previous acute attacks, quite distinct in the memory of the patient from attacks of colic. Some had been previously hospitalized for an acute attack, treated conservatively, and then discharged as recovered. The correlation between the history and the pathologic report was 87.7 per cent correct. The average time lapse between the last definite acute attack and the operation was 2.8 months. Operation as chronic cholecystitis in this group markedly raises the general mortality.

Forty-six cases of perforation of the gall-bladder occurred in so-called chronic cholecystitis, with a mortality of 38 per cent. Nine of these had perforations into the colon. They are included in the 474 cases reported above. The morbidity factor was 2.20, an extremely high index of severe complications. Cholecystostomy for acute cholecystitis was not an unmixed blessing, as even when the patients recovered from the primary operation, subsequent gall-bladder surgery gave a mortality of 20 per cent.

Sixty-eight patients who had a previous cholecystostomy for chronic cholecystitis were operated on again for a recurrence of symptoms. The mortality in this group was 7.4 per cent and the morbidity 1.50. There is no definite manner of determining the

percentage of recurrent symptoms following cholecystostomy in this hospital but that an uncomfortably large number come to subsequent cholecystectomy has been shown by Judd and Priestly,[17] and by Pratt.[18] Barber and Harrison[19] have shown that 54 per cent of those patients with cholecystostomy who could be traced had a recurrence of symptoms. In our group, 77 per cent had recurrent or overlooked stones.

Jaundice in any form added materially to the mortality in chronic cholecystitis. The mortality rate in 254 cases with jaundice at the time of surgery was 13 per cent. The majority of these patients had common duct stones (86 per cent) while the remainder had, in general, a hepatitis. Five patients had biliary cirrhosis (clinical diagnosis).

The factor so well determined in all types of surgery is that of early operation. This factor seems, however, to be reversed relative to chronic cholecystitis where the usual advice is to delay surgery as long as possible. That the follow-up results in cholecystectomy are universally better in patients with moderately long-standing disease cannot be questioned, and will not be discussed here, but that this must be balanced against a rapidly increasing mortality in the older patient is frequently forgotten. Niemeier,[15] and Judd and Priestly,[17] among others, have discussed this problem. It is, however, not so much a problem of the chronologic age of the patient as one of his physiologic age and of the duration of his cholecystic disease. In reviewing the 3,306 operations for chronic cholecystitis, 959 patients were operated upon who had a total duration of symptoms not exceeding two years, who had no evidence of a previous acute attack, and who did not have multiple surgery done. The mean age of this group was 43 years as against a mean age of 47 years in the general group. Therefore, it can be presumed that the age factor was not significant. What seems significant is that only thirteen deaths occurred in this group. The mortality rate here was but 1.35 per cent, in decided contrast to the general mortality in cholecystectomy—3.61 per cent. It would seem logical to conclude that the major factor in the success in this group was the short duration of their symptoms.

ACUTE CHOLECYSTITIS

The 574 cases in this category comprise all the operations on the acute gall-bladder performed during the period of study. No exceptions were made for associated pathology. By using the "pathologic yardstick," we hoped to eliminate most of the error in statistics on the acute gall-bladder. Included, however, are thirty-two cases of undoubted acute cholecystitis in which no pathologic examination was possible because a cholecystostomy was done. They are classified as of pathologically unknown severity but do enter into the results otherwise discussed.

In Table I are found the various operative procedures undertaken for acute cholecystitis with their respective mortalities. It can logically be assumed that those cases having complicated surgery (dochostomy, etc.) were neglected cases, at least neglected as chronic cholecystitis. Their increased mortality can be anticipated. What relationship this increased mortality has to the attitude of immediate intervention or of delay in surgery on the acute gall bladder is an important problem. An attempt is here made to at least indicate where the answer lies.

Table III is a composite of the important facts determined in the study of 574 consecutive operations for acute cholecystitis. Among the total series of biliary tract operations 14.5 per cent were for acute cholecystitis. This is at marked variance with the reports of Mentzer[21] (4 per cent) and Saunders[20] (4 per cent), but much like that of the more recent statistical studies.

Thirty-six per cent of the total acute cases were clinically and pathologically merely acute cholecystitis. The rest, or 64 per cent, were severe cholecystitis, i.e.,

TABLE III
ACUTE CHOLECYSTITIS
Factors Influencing the Mortality and the Morbidity in Surgery in Acute Cholecystitis

Factors	No. of cases	Morbidity Factor	Per Cent Mortality
A) Degree of pathology (Attending pathologist's diagnosis)			
1. Acute cholecystitis	206	1.40	5.85
2. Purulent cholecystitis	117	1.46	9.40
3. Gangrenous cholecystitis	150	1.51	7.33
4. Perforated with abscess	16	2.19	0.00
5. Perforated with peritonitis*	53	2.13	35.85
(B) Complicated cholecystitis (Comparison with acute only)			
1. Acute only	206	1.40	5.85
2. Complicated cholecystitis	368	....	14.10
3. Perforated cholecystitis (known)	69	.15	26.10
(C) Cholelithiasis			
1. Cholecystitis with lithiasis	510	....	12.02
2. Non-calculous cholecystitis	64	....	9.75
(D) Previous evidence of gallbladder disease (Pathological basis)			
1. Acute attack on chronic	433	1.56	12.02
2. Acute with no previous inflammation	141	1.53	7.80
(E) Duration of present illness			
1. Operated within 48 hours of onset.	81 (57 per cent complicated)	1.26	15.10
2. Between 48 and 96 hours (2 to 4 days).	137 (67 per cent complicated)	1.47	5.90
3. Between 4 and 7 days	104 (67 per cent complicated	1.51	7.70
4. Between 7 and 50 days	192 (63 per cent complicated)	1.66	16.71
(F) Duration of the preoperative period of hospital observation			
1. 0–6 hours	128 (63 per cent complicated)	1.51	15.62
2. 6–24 hours	297 (58 per cent complicated)	1.47	7.41
3. 24–48 hours	58 (66 per cent complicated)	1.58	10.35
4. 2–24 days	91 (68 per cent complicated)	1.71	17.59
(G) Preoperative course in hospital (Watchful waiting-all more than 16 hours)			
1. Progressive course	180 (57 per cent of delay cases)	1.73	19.30
2. Static course	100 (31 per cent of delay cases)	1.46	7.00
3. Remissive course	36 (12 per cent of delay cases)	1.65	0.00
(H) Jaundice			
1. Definite past history of jaundice	101	1.64	15.80
2. Jaundice at time of surgery	155	1.68	20.60

* Exclusive of sixteen cases with clinical peritonitis where cholecystostomy was done.

purulent, gangrenous, or perforated. Of the latter group, fifty-three had a free perforation with peritonitis proved pathologically and in sixteen additional cases where cholecystostomy was done, a clinical peritonitis was apparent to the operator. Only sixteen cases had complete localization. That 12.1 per cent proved or 14.5 per cent presumptive peritonitis existed in the operated cases is representative of the experience more recently reported. Branch and Zollinger[22] with 8.6 per cent perforations and Heuer[23] with 20 per cent perforations and gangrene are representative of the recent reports. In spite of these reports, however, Eliason and McLaughlin,[24] and Niemeier[25] believe perforation into the abdominal cavity to be rare. From our observation it would seem that acute cholecystitis is by no means so benign or self-localizing a lesion as was formerly believed.

Heuer,[26] in an exhaustive analysis of 502 cases of perforative cholecystitis, found that the mortality in the various series considered ranged from 33.3 per cent to 52.2 per cent. The mortality rate in this group of sixty-nine proved perforations was 26.1 per cent and for those perforations with peritonitis it was 35.85 per cent. That in this group of perforation with peritonitis there was adequate time to localize may be presumed in view of the fact that the average preoperative duration of illness was eight days.

The presence of stones in the acute gallbladder did not materially increase either the morbidity or the mortality. The incidence of acute cholecystitis is, however, much higher among patients with stones. In operations for chronic cholecystitis only 60 or 65 per cent had stones, whereas 89 per cent of the acute cases had stones. Wolfson and Rothenberg[40] found that 91.9 per cent of their acute cases had stones and believed that non-calculous cholecystitis perforated more readily than calculous cholecystitis. This is counter to the findings here where but one perforation among a

probable eighty-five perforations had no stones.

The rôle of long-standing chronic cholecystic disease in the mortality of acute cholecystitis is still debatable. Seventy-six per cent of all the acute gall-bladders had clinical and pathologic evidence of a previous chronic inflammation. The mortality in this group was 12.02 per cent whereas the mortality in those without evidence of previous disease was 7.8 per cent. Peculiarly, the severity of the infection, on the average, was equal. This suggests that previous cholecystic disease may play a definite rôle in the operative risk of the patient with acute cholecystitis.

Recent literature has dealt mostly with the problem of when, and when not, to operate in acute cholecystitis. The proponents of early operation, notably Heuer,[26] Zinninger,[27] Graham,[28] Pratt,[18] Judd and Grey,[29] Mentzer,[20] Miller,[31] and many others, are increasing in favor. In many instances, however, the defendents of the con servative treatment, notably Pennoyer,[34] Cave,[33] Bass and Bird,[32] Sherwood,[35] and Bruggeman,[36] have had a stronger case statistically. Liedberg,[37] in a recent exhaustive study of the results of ten years of surgery on the gall-bladder at the Lund Surgical Clinic, came to the conclusion that in spite of the apparent operative risk, the acute gall-bladder should be operated on as soon as the patient is reasonably prepared. The "reasonably prepared" may be the crux to the entire problem of the mortality.

Only eighty-one of the 574 cases were operated on within forty-eight hours of the onset of their immediate illness. This small number was not due to a reserve in operating upon the acute gall-bladder, but to the fact that patients did not present themselves for early operation. In this group the mortality was 15.1 per cent or about 40 per cent above the general group. Furthermore, the number of severe cholecystitis cases was less than in the other groups. In spite of this, the group had many fulminating cases including four deaths due to acute hemorrhagic pancreatitis associated with acute cholecystitis.

Those patients operated on from two to four days after the onset of their illness, fared much better. The mortality rate in this group was but 5.9 per cent and the morbidity somewhat lower. This improvement was in spite of a definite increase in the severe cases. After four days of present illness, the patients operated upon had an increasing morbidity and mortality in direct proportion to the duration of the illness.

The surgeon cannot control the stage at which he sees the patient in acute cholecystitis. Therefore the period of hospitalization, preoperatively, has been analyzed. The disproportionate mortality in early surgery is at least in part explained by this, for those patients immediately operated upon on arrival at the hospital (up to six hours) had a mortality of 15.6 per cent and a morbidity of 1.51 with no higher incidence of severe acute cholecystitis. This must be contrasted with a mortality of 7.41 per cent and a morbidity of 1.47 in those cases operated on with the benefit of a period of preparation (six to twenty-four hours). After this period further observation in the hospital only further increases both the mortality and the morbidity until those patients operated on after twelve days of observation had a morbidity of 2 and a mortality of 22 per cent.

Perhaps greater significance should be given the observation that of 316 cases observed sixteen hours or longer in the hospital, 57 per cent had a progression of their symptoms and of their physical findings. Only 12 per cent definitely subsided. In spite of the clinical course, severe pathology was present in all groups. That the mortality rate was 19.3 per cent in those with an apparent progressive lesion, as against no deaths in those definitely subsiding, only emphasizes the futility of the "waiting policy." This study, however, represents only those operated upon and so is open to much error of interpretation. During the period of 1920 to 1930, how-

ever, only forty-two patients were discharged from the Post-Graduate Hospital with a diagnosis of acute cholecystitis who had not had operations.

These results coincide rather remarkably with those published by Graham and Walters[38] as well as those of Tourgoff.[39] Heuer's[4] statement that the immediate clinical course of acute cholecystic disease gives us an index of whether gangrene or perforation will ensue, but that this has no value in a specific case, is especially true in our series.

Even a cursory examination of Table III will suggest a remarkable similarity between acute cholecystitis and acute appendicitis. The effect of delayed surgery on the severity of the infection, the number of free perforations, the ascending mortality and morbidity as the disease progresses, the uncertainty of its course, and the role of preoperative preparation on the mortality, all have a familiar appearance. The major dissimilar factor is the complication represented by the liver and its influence on the risks of surgery on the acute gall-bladder.

This added risk is partially indicated by the results of surgery where there has been a previous jaundice or where there is jaundice present at the time of operation. In 101 patients a history of past jaundice was secured. The mortality rate in this group was 15.8 per cent and the morbidity 1.64. Both are much higher than the general series. Jaundice at the time of surgery increased the hazard exactly 100 per cent in this group of 155 patients. The mortality was 20.6 per cent and the morbidity 1.68. This remarkable increase can only be explained by the pernicious factor of jaundice.

CONCLUSIONS

1. Multiple surgery, in which the gall-bladder is a part, is dangerous and carries a disproportionate mortality and morbidity.

2. Operations on patients with a definite history of previous acute cholecystitis carry a markedly higher risk than operations for chronic cholecystitis. Therefore, the preoperative preparation should be most careful and the prognosis more guarded.

3. Conservative treatment in acute cholecystitis may seem to be immediately successful but eventually may be disastrous.

4. Cholecystostomy, regardless of reason for choice, apparently carries an eventually higher mortality and morbidity.

5. Jaundice, both in the chronic and the acute infections of the gall-bladder, adds nearly a 100 per cent increase to the morbidity and mortality of the patient.

6. The optimum time for surgery on the chronic gall-bladder is within two years of the onset of definite active symptoms, regardless of the chronological age of the patient.

7. Severe cholecystitis as well as perforated cholecystitis is more frequent than is generally believed.

8. Acute calculous cholecystitis is most common. It does not markedly affect the mortality or morbidity in spite of the fact that it predisposes the patient to perforation and peritonitis.

9. Acute cholecystitis upon a chronic lesion definitely increases the operative risk.

10. The lowest mortality and morbidity in acute cholecystitis is found in those patients operated on within four days of the onset of their present illness and after a period of six to twenty-four hours of hospitalization, long enough to prepare them properly.

11. "Watchful waiting" is pernicious as it does not give a true index of the progress of the disease, and makes for delay in surgery with its increased risk.

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A COMPARATIVE STUDY OF THE OPERATIVE MORTALITY IN THE VARIOUS STAGES OF GALL-BLADDER PATHOLOGY*

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THERE were 3,986 consecutive operations on the biliary tract performed at the New York Post-Graduate Hospital from 1920 to July, 1937. The operations were performed by fifty-three attending surgeons. The patients in this group were about equally divided between private and clinic services. The operative mortality and morbidity rate in the series of patients was determined by a study of the clinical history and physical findings, laboratory data, operative findings and procedure, microscopic study and pathologic diagnosis of operative and autopsy specimens, and the postoperative hospital period. Classification of the cases into pathologic groups was made according to the microscopic diagnosis of operative specimens; the macroscopic diagnosis was used only in the few instances in which no operative specimens of tissue were obtained.

A morbidity factor was devised to represent an index of the postoperative complications encountered. A factor of 1 was given each operated case with a relatively normal postoperative course; a factor of 2 where there was a complication of such severity as to prolong the convalescence and yet not ordinarily associated with a mortality; and, a factor of 3 was given to cases with complications potentially fatal. The total factors were divided by the number of cases in the study to give the "morbidity factor."

In making the study in Table 1, the examiner of the case histories was very forcefully impressed by the clinical histories and the pathologic findings of a gradual build-up in the majority of patients with chronic cholecystitis. In the history as well as in the microscopic study of specimens, there was evidence that the gall-bladder patient went through a more or less definite cycle of symptoms and signs of dyskinesia, bile stasis and stone formation, infection with inflammatory changes, loss of gall-bladder function, recurring attacks of

TABLE 1

	Cases	Per Cent Mortality
Operations for Chronic Cholecystitis		
Cholecystectomy*	2,438	3.61
With dochostomy	238	11.34
With secondary surgery	581	13.85
Cholecystostomy	43	30.24
With dochostomy	16	37.50
Total for chronic cholecystitis	3,316	6.40
Operations for Obstructive Biliary Tract Disease		
Cholecystogastrostomy and duodenostomy	52	28.80
Choledochostomy only (after removal of gall-bladder)	39	38.60
Plastic on the bile ducts	5	60.00
Operations for Acute Cholecystitis		
Cholecystectomy*	428	7.47
With dochostomy	89	14.60
Cholecystostomy	45	28.80
With dochostomy	9	33.33
Total for acute cholecystitis (with three other operations)	574	11.97
Total for all biliary tract surgery	3,986	7.7

* With or without appendectomy.

* From the Combined Medical and Surgical Biliary Tract Clinic of the New York Post-Graduate Medical School and Hospital (Columbia University).

acute inflammation that either subsided or led to operation. The advent of jaundice was notable as the common duct became involved in the progress of the disease. From this period on the common duct was seen to undergo changes that might be compared to those previously noted in the gall-bladder, i.e., dilatation, bile stasis, stone formation, and infection with recurring periods of obstruction during which period the pancreas came into the picture more often than in any other stage of the disease. This picture of an advancing process by stages was evident in the history and ulceration of the mucosa were noted in those specimens removed from patients with histories of symptoms of more than two years' duration.

In Table II an attempt is made to classify all the patients operated on for chronic biliary tract disease according to the severity of the disease process as interpreted by the pathologist. The clinical stage, the pathologic physiology, and the pathological diagnosis have been correlated. In each category, the respective mortality and morbidity have been given. The clinical and pathologic evidence seems to indicate

TABLE II

THE CYCLE OF GALL-BLADDER DISEASE AND ASSOCIATED PATHOLOGY
(As seen in patients operated on as chronic cholecystitis)*

Clinical Course	Dysfunction	Stone Formation	Cholecystitis with or without Lithiasis	Severe Cholecystitis with Acute Attacks	Jaundice, Common Duct Dysfunction	Continued Acute Attacks, Jaundice	Jaundice, Active Common Duct Involvement
Associated pathological physiology	Metabolic and dyskinesia	Stasis and lithiasis	Inflammatory changes, loss of concentration power	Ulceration, non-functioning gall-bladder, stones	Dilatation of common duct	Fibrosis, obstruction, perforation	Inflammatory changes, stones, obstruction
Pathologic equivalent	Cholesterosis, "mild" cholecystitis	Mild lithiasis	Thickened gall-bladder, chronic cholecystitis, lithiasis	Severe ulcerative cholecystitis	Dilated common duct (stones)	Hepatitis, perforated gall-bladder, empyema, abd. abscess	Cholangitis, common duct obstruction, stenosis, perforation, pancreatitis
No. of cases in each group	139	820	1213	474	254	70	94
Morbidity factor	1.24	1.22	1.42	1.59	2.04	2.10	unknown
Per cent mortality	1.4	1.34	4.2	11.02	13.0	21.4	26.6

Progressive Disease

* Exclusive of 300 cases with associated gastroduodenal pathology.

in its duration and the incidence of acute exacerbation of symptoms as well as in the corresponding microscopic study of operative specimens in which there was evidence to support the clinical course given. This view is more or less borne out by the microscopic finding of a previous chronic cholecystitis, irregular fibrosis, lymphocytic infiltration and deep scars in those patients operated upon during the acute stages of inflammation. Evidences of previous acute attacks of inflammation, intramural abscess, marked thickening of gall-bladder wall with polymorphonuclear infiltration that cholecystic disease progresses in a relatively definite manner. Meeker,[6] and Meranze, Salzman, and Meranze[7] have recently correlated the results of surgery on the gall-bladder with the severity of the disease. The latter have especially considered the end-results.

In our series of cases the minimum pathology encountered was designated by the pathologists as "mild cholecystitis" and others as "cholesterosis." On reviewing this group of operated cases we have found many to have symptoms suggesting biliary dysfunction. Cole[8] demonstrated that spas-

tic changes in the biliary tract can cause stasis with resulting progressive pathology. He warns, however, against the danger of too readily placing patients in this group. Mackey,[9] in an extensive study on cholesterosis, states that it is "not a specific lesion but rather a histological feature that may occur at random in gall-bladders showing all grades of pathological changes." In our cases cholesterosis was found to be a relatively benign lesion. The mortality rate in the group having minimal lesions was 1.4 per cent in 139 operated cases and the morbidity factor was 1.24.

A large group of patients with stones and minimal thickening of the gall-bladder wall, as well as a number of patients without stone but with inflammatory elements in the submucosa, follow. Whether infection precedes or follows stone formation is subject to much current controversy. The 820 patients comprising this group had generally mild symptoms, though colic was present in a considerable number. This group represents incontroversial gall-bladder disease from the pathologic viewpoint. The mortality rate was 1.34 per cent and the morbidity factor was 1.22.

With a continuation of the disease process the gall-bladder passes into a stage of definite infection and inflammation. There is a loss of function readily ascertainable by clinical tests. The clinical symptoms are increased and on drainage and *x*-ray a pathologic gall-bladder is readily recognized. The largest number of operated cases fall into this group. The mortality rate in surgery on this group of patients increases to 4.2 per cent and the number of severe complications is doubled (morbidity factor of 1.42) in contrast to the preceding group. It is at this stage that the gall-bladder reaches the "critical period" in its disease cycle, i.e., if not removed at or before this time a rapidly ascending morbidity and mortality follow. In addition to progressive chronic inflammation and stone formation, the danger of acute attacks increases. An acute attack is a major catastrophe as it frequently leaves a non-functioning gall-bladder. With the advent of acute attacks in the cycle the mortality rises sharply to 11.02 per cent and the morbidity factor becomes 1.59. In this group there were 474 cases of chronic cholecystitis with pathologic evidence of a previous acute infection. We have found that each succeeding acute attack increases the mortality by approximately 2 per cent.

At this stage, or perhaps earlier, the common duct begins to take over the function of the gall-bladder, with resulting bile stasis, dilatation and stone formation. In addition, gallstones can now more readily enter the common duct from the gall-bladder. Surgery at this stage of the disease had a mortality of 13 per cent and a morbidity factor of 2.04 in the 254 cases operated upon. Continued gall-bladder and common duct disease leads to a total fibrosis and obstruction of the gall-bladder with associated hepatitis, and the potentiality of a chronic perforation of the gall-bladder. The seventy patients operated on at this stage had a mortality rate of 21.4 per cent and a morbidity factor of 2.10. Continued common duct involvement leads to suppurative cholangitis, common duct obstruction, stenosis, pancreatitis, hepatitis and common duct perforation. In ninety-four cases operated at this stage of biliary tract disease the mortality was 26.6 per cent.

It is difficult to place malignancies of the gall-bladder and common duct into this progressive scheme of pathology. In contrast to the findings reported by Boyse and McFetridge[10] and by Jankelson,[11] only nine instances of primary carcinoma of the gall-bladder were found in 3,986 cases, an incidence of 0.2 per cent. They are therefore not considered here.

There were 574 cases of pathologically proved acute cholecystitis among the 3,986 cases operated upon. Tables III and IV represent brief résumés of these cases grouped according to the severity of the pathology with corresponding mortality and morbidity. Contrary to the rule in

chronic cholecystic disease the severity of the acute pathology bore no relationship to the duration of the present illness. Perhaps the outstanding feature of this series of consecutive acute gall-bladders was the increased morbidity and mortality seen in those patients with definite chronic disease before the onset of their acute attack. (Table III.) In those patients with acute

Table III

THE CYCLE OF ACUTE GALL-BLADDER DISEASE
(As seen in 574 pathologically acute gall-bladders)

Clinical Course	History of One or More Acute Attacks or a History of Chronic Cholecystitis			
Pathologic equivalent...	Acute on chronic	Purulent	Gangrenous	Perforated
No. of cases in each group	155	92	108	46
Morbidity factor.....	1.45	1.54	1.42	2.14
Per cent mortality.....	6.4	10.7	8.3	53.7

cholecystitis without evidence of previous disease (Table IV) the mortality was 3.9 per

Table IV

THE CYCLE OF ACUTE GALL BLADDER DISEASE
(As seen in 574 pathologically acute gall-bladders)

Clinical Course	Single Acute Attack—No Definite History of Previous Cholecystitis			
Pathologic equivalent...	Acute	Purulent	Gangrenous	Perforated
No. of cases in each group	51	25	42	23
Morbidity factor.....	1.26	1.25	1.75	2.11
Per cent mortality.....	3.9	4.0	4.8	26

cent and the morbidity factor 1.26, whereas those with previous chronic disease had a corresponding mortality of 6.4 per cent and a morbidity factor of 1.45. Increasing severity of the disease is accompanied by a mathematical progression in the mortality rate and in the morbidity factor. Forty-six cases with proved perforation on a previously chronic process had a mortality rate of 33.7 per cent. Stones were definitely present in 89 per cent of all acute cases operated. Perforation was present in 12.1 per cent examined pathologically and in 14.6 per cent by clinical observation. This frequency of perforation, in which 70 per cent had a peritonitis, is in marked contrast to the reports of Judd,[12] and Niemeier,[13] who believe free perforation to be rare. It is, however, concurred in by Heuer,[3] Pratt,[14] and many others.

Graph 1 shows the mortality and morbidity trends in all cases in which operation was performed for chronic biliary tract disease exclusive of malignancies. Those operated within two years of the onset of definite symptoms had a mortality rate of 2.75 per cent and a morbidity factor of 1.22. This is inclusive of all patients in which a reliable history was obtained. It includes a number of patients who had multiple surgery or had acute attacks. The mortality rate and the morbidity ascends rapidly as the duration of the symptoms increase, until the patients with a definite history of ten years or more had an average mortality of 11 per cent and a morbidity factor of 1.64. Niemeier,[15] and Stevenson,[16] urged earlier operation in recent papers discussing the mortality in chronic biliary tract disease. To be sure Mackey,[17] as well as Judd and Priestly,[18] and many others believe that the longer the duration of the illness and therefore the greater the age of the patient, the better the end result in surgery. Wilson, Lehman, and Goodwin,[19] in a comprehensive study of the follow-up results at the University of Virginia Hospital, found no conclusive evidence of better ultimate results obtained from operation in patients with disease of long standing. We believe that regardless of the validity of the above mentioned reports, the factor of ascending morbidity and mortality in the long duration of symptoms cases is of primary importance.

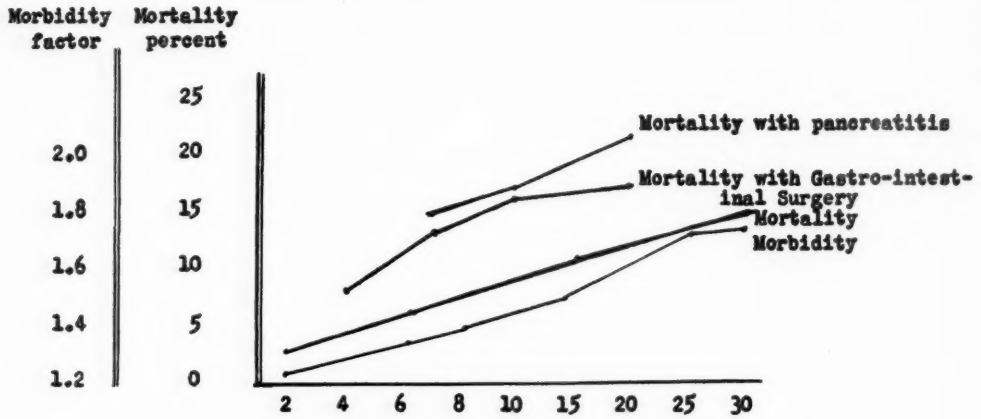
The same general trend of increasing mortality and morbidity as the duration of the symptoms increases is seen in patients operated upon for biliary tract disease who had concomitant pancreatitis and associated gastroduodenal pathology. Pancreatitis was found predominantly in patients having symptoms of seven to twenty years' duration. This group had an average mortality rate of 18 per cent. Gastroduodenal lesions, necessitating surgery, associated with chronic biliary tract disease are found in a much wider range of "symptom age."

Here the average mortality rate is 16.7 per cent. Again a steady increase in risk is noted as the duration of the symptoms is prolonged.

increments regardless of the severity of the infection at the time of operation.

Table v is a brief statistical study of the results in operating on relatively early

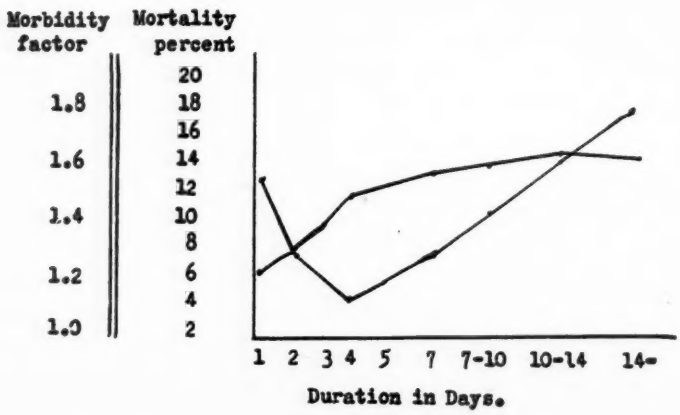
GRAPH I Mortality and Morbidity in Surgery for Chronic Biliary Tract Disease in Relation to the Duration of Symptoms



In Graph II is shown the mortality and morbidity in acute cholecystitis correlated with the duration in days of the present illness. Many factors other than the time element enter into this mortality. They will not be discussed here. It seems apparent

cholecystic disease. All the 1,270 cases were "early" in duration of biliary tract symptoms. There was no absolute correlation between the duration of the biliary tract symptoms and the pathology. There was, however, a general concurrence in that

GRAPH II The Morbidity and Mortality in Surgery on Acute Cholecystitis in Relation to the Duration of the Present Illness.



that the optimun time for surgery is from three to five days after the onset of the attack. On further analysis this proves an unwarranted conclusion. Nevertheless, delay in operating on the acute cases definitely increased both the mortality rate and the morbidity factor by increasing

nearly 80 per cent of the patients in the first two groups in Table II are found in this series. The remaining cases, with short duration of symptoms, were distributed through all stages of biliary tract pathology.

Of the 1,270 cases operated upon in this series, 959 had uncomplicated biliary tract

disease. This "select" group had a mortality of 1.35 per cent and a morbidity factor of 1.22. The addition of an acute attack of cholecystitis, common duct involvement, or gastroduodenal pathology necessitating multiple surgery, increased the mortality rate to 7.1 per cent in 311 cases.

Table V

AN ANALYSIS OF THE RESULTS OF OPERATION IN CHRONIC CHOLECYSTITIS WHEN DEFINITE SYMPTOMS HAVE BEEN PRESENT LESS THAN TWO YEARS

	No.	Deaths	Per Cent Mortality
Uncomplicated cases	959	13	1.35
Complicated*	311	22	7.10
Total operations	1,270	35	2.75

Causes of Death		Major Complications	
Pneumonia	11	Wound infections (severe)	23
Peritonitis	10	Dehiscence	15
Liver death	4	Pneumonia	8
Cardiac failure	4	Thrombophlebitis	5
Operative shock	3	Postoperative hemorrhage	4
Postoperative hemorrhage	2	Pleurisy (effusion)	3
Uremia	1	Cardiac failure	3
		Surgical erysipelas	1
		Peritonitis	1
		Acute parotitis	1

* Those with previous acute attacks or with secondary operations.

The morbidity factor of 1.22 is low in contrast to that found in patients operated upon after prolonged cholecystitis. In spite of this contrast, the incidence of severe complications was relatively high. Wound infections entailing added hospitalization were present in twenty-three instances, or 2 per cent of the operated cases. Dehiscence occurred in fifteen cases, or 1.3 per cent of all operated cases. Pneumonia and thrombophlebitis were next in incidence. The majority of these complications were found in those patients in whom multiple surgery was found necessary. No specific cause for the high incidence of wound infection and dehiscence could be determined.

The mortality rate of 2.75 per cent in this group is in marked contrast to that found in the general series (6.4 per cent) and that found in those with disease giving symptoms over a period of more than ten years (11 per cent). Furthermore it is nearly identical with the general mortality seen in the first three stages of biliary tract disease (3 per cent). Pneumonia and peritonitis lead the causes of death with eleven and ten deaths respectively attributable to these causes. The pneumonia deaths were predominantly in the older patients. Peritonitis had a higher incidence in those cases requiring multiple surgery. Only four of the thirty-five deaths were attributed to "liver death" or hepatic shock. These patients died of the typical syndrome described by Heyd[20] in 1923 and since widely recognized. No autopsies were performed on these patients so that even this diagnosis may be in question. Touroff,[21] in a review of the literature on liver deaths, concludes that it is less frequent than first thought, for often a fulminating intraperitoneal or pulmonary infection escapes detection and mimics liver death. Heyd,[22] in a personally operated series from this hospital, found that 20.5 per cent of the deaths were liver deaths. The low incidence of presumable liver death in the early cholecystitis group is all the more remarkable when contrasted to eighteen of sixty-seven deaths in those patients with symptoms of ten years' or longer duration.

Cardiac failure caused death in four instances. Operative shock was seen in three instances. Two of these patients had, at the time of cholecystectomy, gastric resections for penetrating ulcer. Two patients died of a postoperative hemorrhage. Both were transfused and one was operated on a second time. The hemorrhage in this patient was not due to the release of the cystic artery ligature. The one death due to uremia may well have been due to pancreatic asthenia (Ravdin[13]) as the patient had a common duct drainage.

In an attempt to obviate the diagnostic errors that contribute to the failures of surgery in early cholecystitis, definite indications for surgery on the gall-bladder have been established in the biliary tract clinic of New York Post-Graduate Hospital. These indications are: (1) evidence of stone by means of *x*-ray visualization and

by carefully checked and repeated biliary drainages; (2) evidence of chronic cholecystitis as shown by marked impairment of function of the gall-bladder with evidence of infection in duodenal drainage; (3) common duct involvement as evidenced by the history, biliary drainage, and blood chemistries; (4) a definite acute attack with operation early in the attack or as soon as preparation is completed; and (5) intermittent jaundice indicative of common duct involvement even without other substantiary findings. Those patients who present early metabolic changes or have evidence of dyskinesia without stones are treated medically.

CONCLUSIONS

1. Chronic biliary tract disease, in the main, undergoes a definite cycle of progressive pathologic change.

2. The mortality and morbidity rise directly in proportion to the severity of the pathology, as well as to the duration of the disease as shown by the symptoms.

3. Surgery on the gastroduodenal segment, when performed concurrently with surgery on the biliary tract, carries a prohibitive mortality rate and should therefore be avoided.

4. The end results of cholecystectomy for dyskinesia and early cholecystitis are reputedly poor. These poor results can be avoided by a rigid selection of patients, especially by eliminating those with symptoms due to dyskinesia without stones and with no changes in concentrating function of the gall-bladder.

5. The duration of symptoms in acute cholecystitis, unlike chronic cholecystitis, bear no relationship to the pathologic severity. The mortality and morbidity are, however, favorably influenced by early surgery regardless of the pathology involved.

6. The optimum time for surgery in any given disease of the biliary tract is as early as a positive diagnosis of definite destructive pathology can be made.

(*a*) In chronic disease during the stage of stone formation and minimal inflammation.

(*b*) In acute disease as soon as the patient can be adequately prepared for surgery.

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ACUTE CHOLECYSTITIS*

WITH DISCUSSION OF FACTORS IN MORBIDITY AND MORTALITY

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THE increased interest in the treatment of acute cholecystitis during the past ten years is shown by the large amount of literature on the subject. There has been a trend toward immediate and early surgery but no unanimity of opinion. A surprising similarity is discernible between the recent literature on acute cholecystitis and that on acute appendicitis as seen between 1900 and 1910. At that time there were immediate, early, and late, operators for acute appendicitis, and only after considerable controvery and the accumulation of a tremendous volume of data, was the early operation universally adopted.

No uniformity of opinion on operation for cholecystitis existed among the thirty-one surgeons of the New York Post-Graduate Hospital who operated upon 574 patients presented in this study. A careful analysis of the immediate results and especially of the causes for the failures, in such a series, is a rational basis for conclusions as to the best method of approach.

The purpose of this paper is to review those factors which increased the mortality and morbidity in treatment of acute cholecystitis, and how these were influenced by the time of operation. The factors here considered are: (1) type of operation; (2) degree of pathology present at operation; (3) presence of stones; (4) jaundice at the time of surgery; (5) past history of cholecystic disease; (6) duration of the present attack; (7) period of preoperative preparation; (8) preoperative course; and (9) the eventual result of non-operative treatment.

The data and conclusions presented in this paper were secured from the records of 574 consecutive cases of pathologically proved acute cholecystitis in which operation was performed between January, 1920 and July, 1937. In addition, 474 cases of chronic cholecystitis with pathologic and clinical evidence of a previous acute attack are presented. We believe that the results are those that may reasonably be anticipated in any general hospital of similar prominence and bed capacity.

TABLE 1

OPERATIVE PROCEDURES AND THEIR RESPECTIVE MORTALITY

Operation	No. of Cases		Per Cent Mortality	
Cholecystectomy*	428	517	7.47	8.82
With choledochostomy	89		14.60	
Cholecystostomy*	45	57	28.80	29.63
With choledochostomy†	9		33.33	
Total		574		10.97

* With or without appendectomy.

† Plus three secondary operations.

For the purpose of comparing the immediate postoperative complications a "morbidity factor" has been devised. The morbidity factor is an index of the complications encountered. It is determined by giving an index of 1 to each case with a normal postoperative convalescence, an index of 2 to those with minor complications, and an index of 3 to those with complications potentially fatal. The sum of these indices is divided by the number of cases giving the "morbidity factor."

(1) THE TYPE OF OPERATION

Cholecystectomy vs. Cholecystostomy. A shift in the choice of the operative procedure employed in the treatment of acute cholecystitis can be observed in the recent

* From the Combined Medical and Surgical Clinic for Biliary Tract Disease New York Post-Graduate Medical School and Hospital (Columbia University).

literature. Smith,[1] Leidberg,[2] Smythe and Mason,[3] Stone and Owings,[4] Heuer,[5] Judd and Phillips,[6] and others have dealt with the advantages of cholecystectomy over cholecystostomy. Eliason and Ferguson,[7] as well as Pratt,[8] have said that the gall-bladder, if once infected and inflamed and especially if once acutely so, never will return to a normally functioning state and must frequently be removed at a subsequent operation.

The number of operations performed with their respective mortality rates are given in Table I.

The mortality in cholecystectomy, 90 per cent of all operations, was 8.82 per cent in contrast to a mortality of 29.63 per cent in the fifty-seven cholecystostomies. This disproportionate mortality rate cannot be entirely ascribed to the poor risk of the patients or their relatively more severe pathology, for in a selected group of cases, comparable as to age, severity of infection, and general condition, the mortality rate after cholecystostomy was twice as great as that after cholecystectomy. In addition to this immediate mortality, we found among the cases of chronic cholecystitis studied fifteen patients who had recovered from a cholecystostomy for acute cholecystitis only to have a subsequent cholecystectomy. The mortality rate in this group was 20 per cent, a factor worthy of consideration when determining the efficacy of the two operations.

Choledochostomy in Acute Cholecystitis. It was found necessary to do a choledochostomy in ninety-eight, or 17 per cent, of the 574 cases. Stones were found in the common duct in 82 per cent of those opened for exploration.

In 3,306 operations for chronic cholecystic disease performed during the period of this study, the common duct was explored in only 7.7 per cent of the cases. Failure to consider the common duct in acute cholecystitis frequently leads to operative failure. Judd and Phillips[6] and Beall[9] do not believe that the incidence of common duct disease is high in acute cholecystitis. Beall further believes that the safest policy in acute obstruction is to temporize. Walton,[10] on the other hand, warns that failure to explore and drain the common duct in acute cholecystitis with obstruction may be fatal. Lichty and Zurhorst,[11] as early as 1915 urged that patients with chronic cholecystic disease come to surgery before the common duct is involved. They recognized that acute cholecystitis is relatively frequent in the presence of common duct disease. The mortality for cholecystectomy and choledochostomy was 14.6 per cent in contrast to a mortality of 33.3 per cent where cholecystostomy was combined with choledochostomy. Leidberg,[2] found a similar incidence and mortality rate in the 556 cases of acute cholecystitis operated at the Lund clinic.

(2) PATHOLOGY

The microscopic pathologic diagnosis of acute cholecystitis has been used in compiling this series of cases to avoid the varying interpretation and terminology which thirty-one independent surgeons placed on the macroscopic gall-bladder pathology. Thirty-two patients had no tissue removed upon which a microscopic pathologic diagnosis could be made. This small group is

TABLE II

MICROSCOPIC PATHOLOGIC CLASSIFICATION OF ACUTE CASES OPERATED UPON WITH THE MORTALITY AND MORBIDITY SEEN IN EACH GROUP

Pathologic Diagnosis	No. of Cases	Per Cent of Total Series	Per Cent Mortality	Morbidity Factor
Acute cholecystitis	206	36.0	5.85	1.40
Purulent cholecystitis	117	20.4	9.40	1.46
Gangrenous cholecystitis	150	25.9	7.33	1.51
Perforated with abscess	16	2.8	0.00	2.19
Perforated with peritonitis	53	9.2	35.85	2.13
No pathologic report	32	5.7	34.40	
Total	574	100.0	10.97	1.55

included because clinically and macroscopically they unquestionably represented acute cholecystitis. The microscopic pathologic report and the macroscopic diagnosis as made by the surgeon coincided in 92.3 per cent of the cases.

A summary of the pathology encountered in this series is presented in Table II. The cases are grouped as to the severity of the infection and their respective mortality and morbidity.

Acute Cholecystitis. The pathologic diagnosis of "acute" cholecystitis includes all acute inflammations of the gall-bladder which have not progressed to acute empyema, gangrene, or perforation. Thirty-six per cent of the cases belong to this pathologic group. These cases were, in many instances, early cholecystitis, i.e., early in duration of their acute symptoms. Thirty-four per cent of these "acute" cases were, however, seen after a week's illness. Twenty per cent of the total "acute" cases were non-calculous. Non-calculous cholecystitis predominated in patients with a long duration of the immediate illness.

The mortality rate in "acute" cholecystitis was 5.85 per cent. This was the lowest mortality rate found in our acute series for any pathologic group. In spite of the relatively benign pathology of these cases the mortality was found predominantly in those which had common duct involvement. The morbidity factor in this group was 1.40. This is a rather high index of complications but one lower than in any other group.

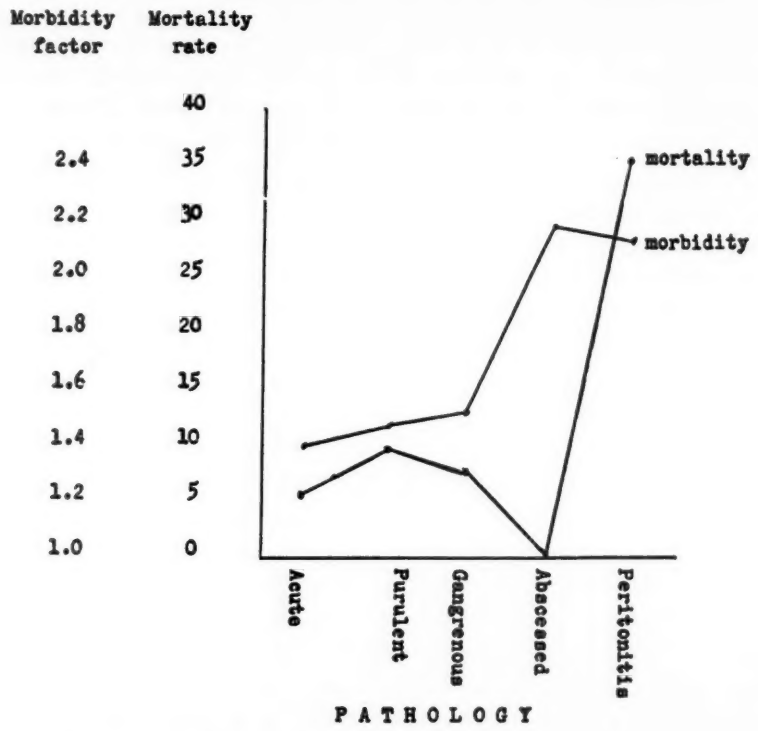
Purulent Cholecystitis. Acute empyema or purulent cholecystitis was found in 20.4 per cent of all acute gall-bladders operated upon. It was most frequently found associated with an acute obstruction at the cystic duct. Mentzer[12] believes that the initial lesion is an acute infection which, through its associated edema and the efforts of the gall-bladder to empty, causes an acute obstruction. This is our experience as a stone was found impacted at the ampulla or the cystic duct in 89 per cent of all patients with acute purulent cholecystitis. The mortality rate in acute empyema was 9.40 per cent and the morbidity factor 1.46.

Gangrenous Cholecystitis. Gangrene of the gall-bladder wall was seen in 25.9 per cent of all the cases. This is a higher incidence than is usually reported. Many of these were seen relatively early in the acute attack and they would, most likely, have perforated upon further operative delay. Stones were present in 94 per cent of all gangrenous gall-bladders. The mortality rate of 7.33 per cent found in this group is not high when the severity of the disease is considered. It is lower than that for acute empyema as the duration of the acute illness was generally much shorter. The morbidity factor was 1.51, a slight increase over that seen in empyema.

Perforated Cholecystitis with Abscess. Sixteen cases of perforation occurred, with localization into a well defined abscess. This represents a surprisingly small number of the total acute cases (2.8 per cent). The serious nature of a perforated gall-bladder is recognized when one considers that only 23 per cent of the proved perforations were localized at the time of operation. Stones were present in the gall-bladder in each instance save one and in this case a common duct stone was found. This low incidence of abscess formation is in marked contrast to that reported by Eliason and McLaughlin,[13] Niemeier,[14] Judd and Parker,[15] Pennoyer,[16] and others. These authors are generally of the opinion that acute perforated cholecystitis tends to localize in the majority of instances. McClure and Altemeier,[17] in an excellent study of acute perforated appendicitis, found in 46.43 per cent of their cases that localization was present at operation. From our study, it is clear that the gall-bladder is less likely to localize than is the acute appendix. The lower incidence of abscess cannot be attributed to early surgery, for the average duration of the acute symptoms in the abscess group was 9.8 days and that of the peritonitis group (exclusive of pancreatitis) was 9.3 days.

No deaths occurred in the sixteen cases after operation for abscess. Cholecystectomy was done in all but one. The number of severe complications was great, for this group had a morbidity factor of 2.19.

GRAPH I The Mortality and Morbidity in Acute Cholecystitis in relation to the severity of the pathological process.

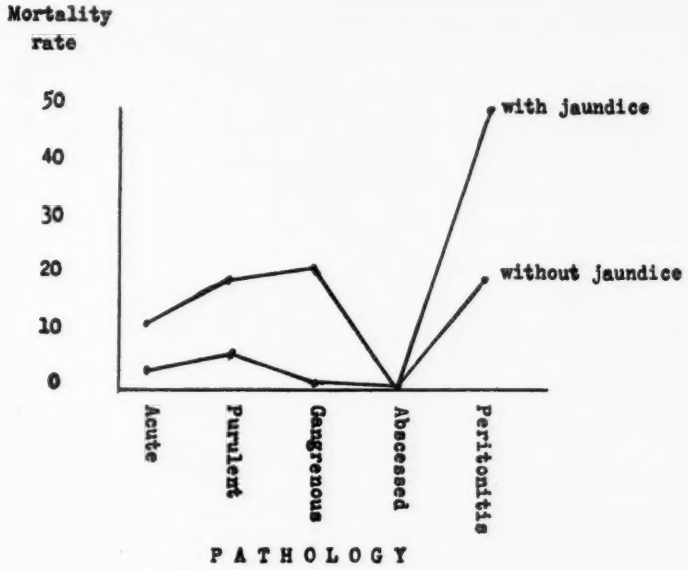


Perforation with Peritonitis. Acute perforated cholecystitis with peritonitis was found in 9.2 per cent of all patients operated on for acute cholecystitis. Diffuse or generalised peritonitis was found in 60 per cent of these, having been limited in the rest by adhesion of the omentum and the colon. This incidence is quite comparable to that seen in delayed treatment for acute appendicitis. The incidence of peritonitis in our cases is much higher than that reported by Eliason and McLaughlin[13] (1.8 per cent), Niemeier[14] (0.7 per cent), Smith[18] (2.3 per cent), Mitchell[19] (1.2 per cent), and Leidberg[2] (2.6 per cent). More recent reports by Boyce, Veal, and McFetridge[20] (5.8 per cent), Branch and Zollinger[21] (8.7 per cent), and Heuer[5] (21 per cent for gangrene and perforation) show that the incidence of perforation is much higher than formerly thought.

The mortality rate for free perforation of the gall-bladder was 35.85 per cent and the morbidity factor was 2.13. When all sixty-nine cases of perforation are considered, the mortality rate is 26.1 per cent. Such a severe mortality places the gall-bladder high among the potentially fatal acute conditions of the abdomen. Heuer[22] collected 502 perforations of the gall-bladder from the literature. He found that the mortality rate in this group varied from 33.3 per cent to 52.2 per cent. During the last five years covered by this study there has been a marked decline in the mortality rate due to perforated acute cholecystitis with peritonitis (22 per cent). In this period the number of perforations have decreased slightly though the total operations for acute cholecystitis have increased. We believe that both the decrease in incidence and the decrease in mortality are due to better preparation and earlier operation.

The increase in mortality and morbidity, as the severity of the infection increases, which was experienced in our series, is shown in Graph I.

GRAPH II The influence of jaundice on the mortality in Acute Cholecystitis



(3) CHOLELITHIASIS

Stones were present in the gall-bladder in 89 per cent of the acute cases which came to operation. The general mortality of those with stones was higher than in non-calculous acute cholecystitis (11.12 per cent as compared with 9.75 per cent). As stones were present in all cases with perforation a much higher mortality was anticipated. (Table III.) Actually, acute non-calculous cholecystitis had a higher mortality in the presence of similar pathology. Wolfson and Rothenberg[23] believe that the general severity of acute non-calculous cholecystitis is greater than that of calculous cholecystitis. Contrary to our findings, however, they believe that non-calculous cholecystitis has a higher incidence of perforation.

TABLE III
A COMPARISON OF THE MORTALITY AND MORBIDITY IN ACUTE CALCULOUS AND NON-CALCULOUS CHOLECYSTITIS

	No. of Cases	Per Cent of Total	Per Cent Mortality	Morbidity Factor
Cholecystitis with stones	510	89.0	11.12	1.57
Cholecystitis without stones	64	11.0	9.75	1.40

(4) JAUNDICE

Jaundice was present in 155 patients operated upon for acute cholecystitis. The pathology encountered in the jaundiced patients was generally more severe than that in the general group. Fifteen per cent of all cases with jaundice had diffuse peritonitis. Common duct involvement was present in sixty-seven of these jaundice patients so that either a choledochostomy or a cholecystostomy was performed. The mortality rate in patients with jaundice was nearly twice that for the general series (20.6 per cent); the morbidity factor was 1.68, a very high incidence of severe complications.

The influence of jaundice on the mortality in acute cholecystitis is graphically represented in Graph II. The lower line is the mortality curve in those cases without jaundice and the upper line is that of cases with jaundice. In each pathologic classification, other than abscess, the presence of

jaundice added greatly to the severity of the prognosis.

(5) PAST HISTORY

The mortality and morbidity in acute cholecystitis is adversely influenced by a previously existing chronic cholecystitis. Pathologic evidence of a preëxisting chronic cholecystitis was found in 75.5 per cent of the acute cases. They are classified as "acute on chronic" cholecystitis. The clinical history coincided with this pathologic finding in 89 per cent of the cases where there was a reliable history. The average duration of the chronic cholecystitis symptoms was nine years. It was in this group that common duct involvement was most frequently found associated with an acute cholecystitis.

A comparative study of the mortality as seen in cases operated with an "acute on chronic" pathologic diagnosis and those with an acute "first attack" pathologic diagnosis is shown in Table IV. The mortality rate was definitely higher in each pathologic group in which the patients had evidence of a previous chronic cholecystic disease. This increased mortality is seen in spite of the fact that the severity of the infection was slightly greater in the "first attack" cases. Prolonged liver damage due to neglected chronic cholecystitis is the probable cause for the added mortality. Niemeier[24] and Graham[25] stressed the factor of long standing chronic cholecystic disease as a cause for acute cholecystitis. They found that "acute on chronic" disease is frequently associated with common duct involvement, jaundice, cholangeitis, and liver damage. Niemeier[24] found the mortality rate higher in surgery on the "acute on chronic" cases.

The morbidity was generally higher in those cases of acute cholecystitis which indicated pathologic evidence of a previous chronic cholecystitis or a previous acute attack. (Table v.)

TABLE IV
THE COMPARATIVE MORTALITY IN "ACUTE ON CHRONIC" CHOLECYSTITIS AND IN THOSE OPERATED ON DURING THEIR FIRST ATTACK

Pathology	"Acute on Chronic" No. Cases	Per Cent Mortality	"First Attack" No. Cases	Per Cent Mortality
Acute only	155	6.4	51	3.9
Purulent	92	10.8	25	4.0
Gangrenous	108	8.3	42	4.8
Perforated with abscess	11	0.0	5	0.0
Perforated with peritonitis	35	37.1	18	33.3
Unknown pathology	32	34.4		
Total	433	12.02	141	7.80

TABLE V
THE COMPARATIVE MORBIDITY IN "ACUTE ON CHRONIC" CHOLECYSTITIS AND IN PATIENTS OPERATED ON DURING THEIR FIRST ATTACK

Pathology	"Acute on Chronic" No. Cases	Morbidity	"First Attack" No. Cases	Morbidity
Acute only	155	1.45	51	1.26
Purulent	92	1.54	25	1.25
Gangrenous	108	1.42	42	1.75
Perforated with abscess	11	2.10	5	2.40
Perforated with peritonitis	35	2.15	18	2.00
Unknown pathology	32			
Total	433	1.56	141	1.53

TABLE VI
A COMPARISON OF THE COMPLICATIONS, PERFORATIONS, MORTALITY AND MORBIDITY ENCOUNTERED IN 514 CASES IN WHICH THE DURATION OF THE PRESENT ILLNESS WAS KNOWN

Duration of the Immediate Illness	No. of Cases	Per Cent Complicated	Per Cent Perforated	Per Cent Mortality	Morbidity Factor
Up to 48 hours	81	56.8	14.8	11.1	1.26
2–4 days	137	67.2	6.0	5.9	1.47
4–7 days	104	67.0	12.3	7.7	1.51
7 or more days	192	63.6	17.2	16.7	1.66
Totals	514	64.0	12.4	10.97	1.51

(6) DURATION OF THE IMMEDIATE ILLNESS

When should acute cholecystic disease be operated upon? Around this question has revolved most of the controversy on the treatment of acute cholecystitis. Cave,[26] in a very logical paper, has divided the present surgical opinion into three groups, namely the immediate, early, and delayed operators. He pointed out that none of the operators taking a definite stand on when to operate have done so to the exclusion of all other methods and that therefore the time of operation is dependent upon the judgment of the operator. This is a rational stand and one generally adhered to. In the main, however, surgical thought is divided into a rapidly increasing group of "early" operators and a diminishing group of "conservative" operators.

Foremost among the exponents of early operation are Heuer,[5,22,27] Glenn,[28] Miller,[29] Smith,[30] Graham and Waters, [31] Touroff,[32] Mentzer,[12] Leidberg,[2] and Walton.[10] These are but a few of many "early" operators. The conservative operators include Cave,[26] Pennoyer,[16] Behrend,[33] Smyth and Mason,[3] Bass and Bird,[34] Branch and Zollinger,[21] Bruggeman,[35] and Sherwood.[36] The attitude of an increasing number of surgeons is summed up very well by Heyd,[37] who states, "for the physician to counsel waiting in acute disease is to participate in a surgical gamble that 'under a regime of starvation and local applications, an ascending phase of pathological change will become arrested.' This is distinctly a gamble with the odds against him."

A summary of the effect on the morbidity and mortality in acute cholecystitis of the duration of the present illness is shown in Table VI. The history was reliable in only 514 cases and these alone are presented. In this series there are patients operated upon immediately, early, and late in the course of the disease. The study should, therefore, be of some value in determining the optimum time for surgery. The mortality rate in the eighty-one cases in which operation was carried out within two days of the onset of their acute symptoms was 11.1 per cent. This is nearly twice that found in the next succeeding group. This high mortality is an apparent argument against early surgery. On further study, eleven of these early cases proved to be fulminating peritonitis, including seven with acute hemorrhagic or suppurative pancreatitis. Eight of these eleven died. If these cases were to be eliminated from this series the mortality rate in patients operated on within forty-eight hours of onset would have been 2.9 per cent. The morbidity factor in these cases was 1.26, an extremely low incidence of severe complications when contrasted to those encountered in delayed surgery.

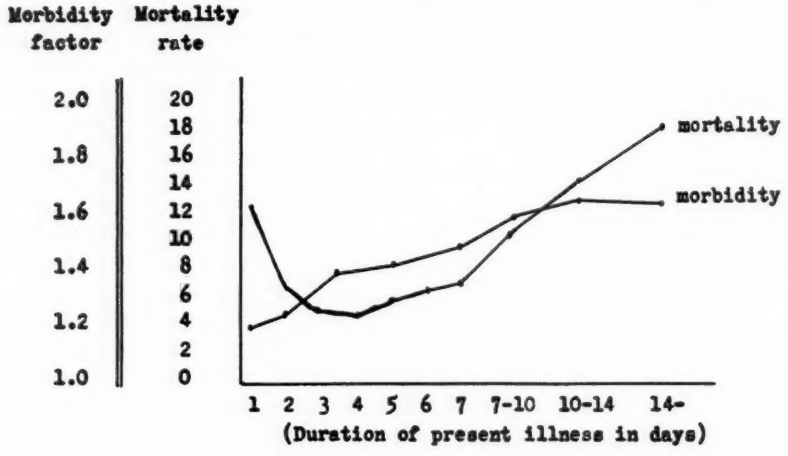
One hundred and thirty-seven patients were operated upon during the period of two to four days following the onset of acute symptoms with the low mortality of 5.9 per cent. More of these cases had complicated disease (67.2 per cent) than did those operated earlier in their acute attack, but the incidence of fulminating disease was much lower. With the exclusion of the fulminating cases from the earlier group, the mortality in those operated on two to four days after the onset of symptoms was nearly twice that of the first group.

In 104 cases operation was done between four and seven days after the onset of their acute symptoms with a mortality of 7.7 per cent and a morbidity factor of 1.51. In spite of a constant distribution of pathology as contrasted to the earlier operation the morbidity and mortality increased.

The morbidity and mortality increased steadily with longer delay in operating. The largest group to be operated upon were those with symptoms which had gone on for more than seven days. In this group the mortality rose to 16.7 per cent. Actually it was a graduated increase in mortality; those patients operated on two weeks or longer after the onset of their acute attack had a mortality of 18 per cent. The morbidity of this group rose similarly to a factor of 1.66. This represents a very high index of complications in view of the

fact that these patients had, as a group, less severe pathology than the preceding groups.

GRAPH III The mortality and morbidity in Acute Cholecystitis in relation to the duration of the present illness.



The morbidity and mortality trends in relationship to the delay in operating on acute cholecystitis are represented in Graph III.

(7) PREOPERATIVE PREPARATION

The determination of when to operate in the course of disease is all too frequently beyond the control of the surgeon. General practitioners have not, in the past, hospitalized patients with acute cholecystitis until the progress of the disease had become alarming. For this reason an analysis has been made of the influence of the preoperative preparation on the mortality and morbidity of acute cholecystitis. The influence of the length of preoperative hospitalization on the mortality and morbidity of acute cholecystitis is shown in Table VII.

TABLE VII
THE MORTALITY AND MORBIDITY IN ACUTE CHOLECYSTITIS IN RELATION TO THE LENGTH OF PREOPERATIVE HOSPITALIZATION

Duration of Observation Period	No. of Cases	Per Cent Perforated	Per Cent Mortality	Morbidity Factor
0–6 hours.....	128	10.0	15.6	1.51
6–24 hours.....	297	13.1	7.4	1.47
24–48 hours....	56	8.6	10.35	1.58
2–24 days.....	93	12.8	17.60	1.71
Total.....	574	12.1	10.97	1.55

In the 128 cases in which operation was done immediately upon admission to the hospital (up to six hours) the mortality was 15.6 per cent and morbidity 1.51. This group represents a number of fulminating cases as well as four with acute hemorrhagic pancreatitis. The high mortality cannot be explained on this basis alone for even when these are removed from the group the mortality in immediate surgery is 12 per cent. Judd and Grey,[38] Smith[30] Mentzer[12] and others have warned against immediate surgery without preparation. It is apparent from the high mortality rate in operating upon patients in an emergency, that this is a pernicious practice. In every pathologic type, other than gangrene, the mortality rate was higher in immediate surgery.

The 297 patients operated on after a period of preparation (six to twenty-four hours) had a mortality of 7.4 per cent and a morbidity factor of 1.47. The general index of pathologic severity was equal to that seen in other groups. These patients were hospitalized long enough for reason-

able preparation and the estimation of their probable surgical risk, and yet not so long as to allow a marked progress in the disease. Eliason and Erb[39] have shown the decrease in mortality and morbidity in surgery on the biliary tract, including acute cholecystitis, which has followed adequate preparation by means of glucose, transfusions, etc. This factor is well demonstrated in this series of cases.

The fifty-six patients operated on during their second hospital day did not fare well. They had a mortality of 10.35 per cent and a morbidity factor of 1.58. A small but definite increase in the severity of the pathology was noted though the incidence of peritonitis decreased. An additional twenty-four hours of preparation had not decreased the operative risk in these cases.

In the ninety-three cases in which delayed operation was practiced we encountered the highest mortality and morbidity in the series (17.6 per cent and 1.71 respectively). In spite of hospital care and observation this group showed progressive pathology. The penalty for delayed operation was a 250 per cent increase in the mortality and a 50 per cent increase in the severe complications.

Table VIII

AN ANALYSIS OF THE CLINICAL COURSE OF DISEASE IN 316 CASES OBSERVED IN THE HOSPITAL FOR EIGHTEEN HOURS OR MORE

Apparent Clinical Course of Disease	No. of Cases	Per Cent of Total	Per Cent Mortality	Morbidity
Progressive....	180	57	19.3	1.73
Static.....	100	31	7.0	1.46
Remissive.....	36	12	0.0	1.65

(8) PREOPERATIVE COURSE OF THE DISEASE

"Watchful waiting" was long considered good therapy in acute cholecystitis. Smyth and Mason,[3] Smith,[1] Cave,[26] and Pennoyer[16] are convinced that observation of the clinical course will determine the necessity for surgical intervention. The "early operators" are unanimous in condemning this procedure. Touroff,[32] Heuer[5] and Mitchell[19] noted that the symptoms of acute cholecystitis may subside and yet the lesion may progress. This has been our experience.

A résumé of the preoperative course of 316 cases observed in the hospital for more than eighteen hours is given in Table VIII. Fifty-seven per cent showed evidence of a progressive lesion with increasing toxemia, temperature, pulse, physical and laboratory findings. In this group the mortality was 19.3 per cent and the morbidity factor 1.73. Waiting to see whether or not the acute infection would subside was a gamble not justified by the results. During this period, to determine the course of the disease, a considerable number perforated and twenty-seven had a peritonitis when operated on. The mortality in this group with delay and then surgery for peritonitis was 48.1 per cent.

In 100 cases it was impossible honestly to determine the progress of the disease. It is probable that most of these cases would have subsided. In spite of lack of evidence of a progressive lesion, six patients had a diffuse peritonitis when operated on.

Only thirty-six patients were operated upon with a course in the hospital that definitely indicated a subsiding or subsided lesion. There were no deaths in this group though fourteen had severe cholecystitis. An additional forty-two patients were discharged from the hospital without surgery but with the diagnosis of an acute cholecystitis. They had all subsided clinically.

(9) NON-OPERATIVE TREATMENT

What happens to patients with acute cholecystitis who do not come to surgery? How many are subsequently operated on during an acute attack or as chronic cholecystitis? What are the mortality and the morbidity in these cases? The eventual fate of all patients with acute cholecystitis can only be determined when these questions are answered. In our series it was

TABLE IX

ANALYSIS OF 474 PATIENTS OPERATED UPON AS CHRONIC CHOLECYSTITIS WHO HAD CLINICAL AND PATHOLOGIC EVIDENCE OF A FORMER ACUTE ATTACK

Pathologic diagnosis of former acute attack	474
Average total duration of chronic history	9 years
Average time since last acute attack (History and clinic record)	2.8 months
Mortality rate	11.20 per cent
Morbidity factor	1.59

impossible to answer them, but some information has been gained in analyzing 474 cases of chronic cholecystic disease in which pathologic findings were suggestive of a former acute infection. (Table IX.)

The pathologic evidence taken as suggestive of a former acute infection was by no means conclusive. It consisted of: (1) evidence of scar tissue penetrating the muscular layer of the gall-bladder; (2) chronic ulceration; (3) chronic empyema; and (4) chronic perforation of the gall-bladder. Further evidence was found in that in 87.7 per cent of these cases there was a history suggestive of an acute attack.

The mortality rate and the morbidity factor were somewhat higher in these cases than in those operated for acute cholecystitis. They were remarkably higher than these same factors in the 3,306 cases of chronic cholecystitis of which they were a part. The general mortality of all cases of chronic cholecystitis was 6.4 per cent and the morbidity factor 1.31.

CONCLUSIONS

1. The results of cholecystectomy for acute cholecystitis are superior to those of cholecystostomy regardless of the severity of the disease encountered.

2. Common duct disease is frequently present with acute cholecystitis. When present, it adds greatly to the operative risk.

3. The mortality and morbidity increase in direct proportion to the severity of the pathology.

4. Perforation of the acute gall-bladder with peritonitis has as high an incidence as has peritonitis in acute appendicitis. The peritonitis does not tend to localize, as is generally believed.

5. Cholelithiasis, present in acute cholecystitis, predisposes toward perforation.

6. Jaundice, regardless of cause, doubles the operative risk in acute cholecystitis. It is usually found in acute disease following upon neglected chronic disease.

7. "Acute on chronic" cholecystitis carries a higher mortality and morbidity than does "acute-first attack" cholecystitis. The mortality in the former is probably proportionate to the severity of the preëxisting chronic disease.

8. Furthermore, the mortality and morbidity of surgery on the acute gall-bladder is directly proportionate to the duration of the acute attack. (This exclusive of cases with acute hemorrhagic pancreatitis.)

9. Immediate surgery, without preoperative preparation, carries a high mortality.

10. "Early" surgery, after a period of preparation not exceeding twenty-four hours, has the lowest mortality and morbidity.

11. Delayed surgery, after a period of hospitalization of two days or longer, has a mortality rate and a morbidity in direct proportion to the added delay.

12. "Watchful waiting" is a gamble unjustified by the increased morbidity and mortality attendent on delay.

13. The clinical course of the patient gives no index to the pathological severity of the disease.

14. What appears to be an initially successful conservative treatment in acute cholecystitis may finally result in a higher mortality and morbidity for the patients than is found in those operated upon during the acute attack.

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PERFORATED CHOLECYSTITIS*

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SURGERY on the acute gall-bladder has long been disputed because of a difference of opinion as to the incidence of the severe complications attending this acute abdominal infection. There are, therefore, two opposing methods of treating acute cholecystitis: (1) operating as soon as the diagnosis is made; and (2) observing the course of disease and operating only when intervention is imperative. They arise from a difference of opinion first, as to the incidence of perforation and peritonitis in acute cholecystitis, and second, as to the seriousness of a perforation. The belief has been generally held that perforation was unusual in acute cholecystitis and further that when it occurred localization was the rule.

Eliason and McLaughlin[1] Branch and Zollinger,[2] Smith,[3] Cave,[4] and Pennoyer[5] are but a few of those who believe that perforation either is unusual or generally localizes when it does occur. Saunders,[6] Heuer,[7] Heyd,[8] Stone and Owings,[9] and Walton[10] are among those who have found that perforation, without localization, is frequent in acute cholecystitis. Dependent, then, on these fundemental beliefs is the choice of early surgery, as now practiced in acute appendicitis, or delayed, late surgery as in acute salpingitis.

We are presenting a series of 115 perforations of the gall-bladder encountered in the 3,880 cases of acute and chronic cholecystitis operated on at the New York Post-Graduate Hospital during the years 1920 to July, 1937.

INCIDENCE OF PERFORATED CHOLECYSTITIS

A résumé of the cases of perforated cholecystitis operated upon in this series is given in Table 1. The incidence in percentages, the associated mortality, and the index of severe complications (morbidity factor) are also given. The morbidity factor is determined as follows: A factor of 1 is given each case with a normal convalescence. A factor of 2 is given cases with mild complications. A factor of 3 is given cases with a complication potentially fatal. The means of these equals the "morbidity factor."

TABLE 1

THE INCIDENCE OF PERFORATED CHOLECYSTITIS

(In patients who have recovered from acute attack and are subsequently operated on and the incidence in operated acute cholecystitis.)

Pathology (Microscopic Diagnosis)	No. in Series	Per Cent Perforated	Per Cent Mortality	Morbidity Factor
Chronic cholecystitis (previous acute attack)	474	....	11.2	1.59
"Chronic" perforated cholecystitis	46	9.7	19.5	2.20
Acute cholecystitis	574	....	10.97	1.55
Acute perforated cholecystitis	69	12.1	26.1	2.13
Total cases of perforated cholecystitis	115	11.0	24.3	2.16

The incidence of perforation in all cases operated for cholecystic disease was only 3 per cent. This is not a true index of its frequency for perforation is rare or unknown in true chronic cholecystitis. Therefore the reports from various clinics will differ depending on the type of cases. As a typical instance Judd and Phillips[11] report an incidence of perforation of 0.9 per cent. They recognized the fact that severe acute disease was not generally encountered at the Mayo Clinic. When only the acute cases are considered, together with those with presumptive previous acute

* From the Combined Medical and Surgical Clinic for Biliary Tract Disease, New York Post-Graduate Medical School and Hospital (Columbia University).

disease, the incidence of perforation is 11 per cent. (Table I.)

"CHRONIC" PERFORATED CHOLECYSTITIS

The 46 patients with "chronic" perforated cholecystitis comprising this series were, on the whole, operated on for chronic cholecystitis. They all had both pathologic and historical evidence of previous acute attacks an average of 2.8 months before surgery. It is probable that the perforation, in the vast majority, took place at this time. They are typical of the "hidden perforations" discussed by Touroff[12] and Mitchell.[13] Either perforation was not recognized or the attending peritonitis became self-limited. In general, however, the clinician did not recognize that perforation had taken place.

The conservative treatment of acute cholecystitis is attended by a serious risk of perforation, as is well illustrated in this series of 474 cases with their forty-six perforations. The mortality rate was slightly higher than in operation for acute cholecystitis, in spite of the fact that many of the latter cases represented surgical emergencies. In those cases where perforation and localization had occurred, the mortality rate was 19.5 per cent on subsequent operation, and the morbidity was very high.

Six patients with "chronic" perforations were operated on for bowel obstruction. In each instance the gall-bladder had perforated into the colon, and adhesions, edema, and stones caused the obstruction. In three a cholecystoduodenal fistula existed. Common duct obstruction was found in eighteen cases.

Death in "chronic" perforated cholecystitis was due primarily to peritonitis and "liver death." The first cause is that inherent in the technical difficulties involved in removing a chronic, perforated gall-bladder, especially if a common duct obstruction or the perforation of an adjoining viscus is a complicating factor. The second cause, liver death, is due to the severe liver damage so often found in neglected cholecystitis. Colp and Ginsberg,[14] in an excellent study of the cause of death following surgical diseases of the biliary tract, found seven patients with a suppurative peritonitis following operations for chronic cholecystitis. They also found a surprising number of deaths in which surgery was not done or where palliative measures only were attempted in moribund patients.

TABLE II

A CLASSIFICATION OF CASES WITH PERFORATION ON THE EXTENT OF THE ASSOCIATED PERITONITIS

Pathology	No. of Cases	Per Cent Mortality	Morbidity
Perforated with abscess (localized)	16	0.0	2.19
Perforated peritonitis (local and general)	46	30.5	2.13
Perforated—peritonitis and acute pancreatitis	7	71.4	
Total acute perforation	69	26.1	2.15

ACUTE PERFORATED CHOLECYSTITIS

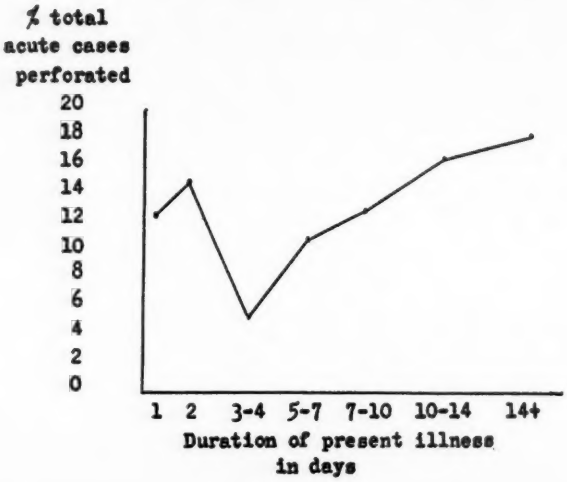
Twelve per cent of all patients operated upon for pathologically proved acute cholecystitis had perforations. A number of very severe fulminating cases were in this group, including seven of acute suppurative or hemorrhagic pancreatitis. The incidence of perforation generally found in our series is comparable to that in acute appendicitis (Keyes[15]). A brief résumé of the acute perforations encountered in 574 cases of acute cholecystitis is given in Table II.

Perforation with Abscess. Only 23 per cent of all proved acute perforations had localized at the time of operation. Stones were present in each instance. It is evident that localization was not the rule in our series. The low incidence of abscess formation cannot be attributed to early surgical interference, for the average pre-hospitalization duration of the acute illness in abscess cases was 9.8 days and in the

peritonitis cases was 9.3 days (exclusive of those with pancreatitis).

There were no deaths among those operated on for an abscess in spite of the fact that cholecystectomy was done in fifteen of the sixteen cases. It is probable that the cause for the success in these cases lies in the cause for localization, *i.e.*, the low virulence of the organisms involved. The morbidity was very high. Ileus, thrombophlebitis, pneumonia, and wound infection were responsible for most of the severe complications.

GRAPH I Incidence of Perforation in Acute Cholecystitis in relation to the duration of the P.I.



Perforation with Peritonitis. Fifty-three patients, or 77 per cent of all cases with perforation, had peritonitis at the time of operation. Sixty per cent had a diffuse or generalized peritonitis, while in the remainder inflammation was localized by the omentum and colon. This high incidence of peritonitis in acute perforation is substantiated by the findings of Heuer,[7], Boyce, Veal and McFetridge,[16] Pratt,[17] Glenn,[18] and others. The continual menace of a diffuse peritonitis in acute cholecystitis is thus apparent. McClure and Altemeier,[19] in a recent comprehensive report on perforated appendicitis, found peritonitis in only 53 per cent. Comparatively speaking, the danger of a diffuse peritonitis, as seen in our series, is as great in acute cholecystitis as that generally reported in acute appendicitis.

Were the patients operated upon too early? Would localization have followed, if more time had been allowed? These questions are partially answered in Graph 1 which shows the incidence of perforated cholecystitis among the total cases of acute cholecystitis in which operation was done. Only the cases with a reliable history are included. There is a high incidence of perforation during the first two days of illness; it then drops and increases again in proportion to the delay in operating. Perforation occurred as late as fifty days after the onset of the acute symptoms, although in the majority of cases it was

TABLE III
ANALYSIS OF SIXTY CASES OF ACUTE PERFORATED CHOLECYSTITIS ON THE BASIS OF THE TIME OF OPERATION

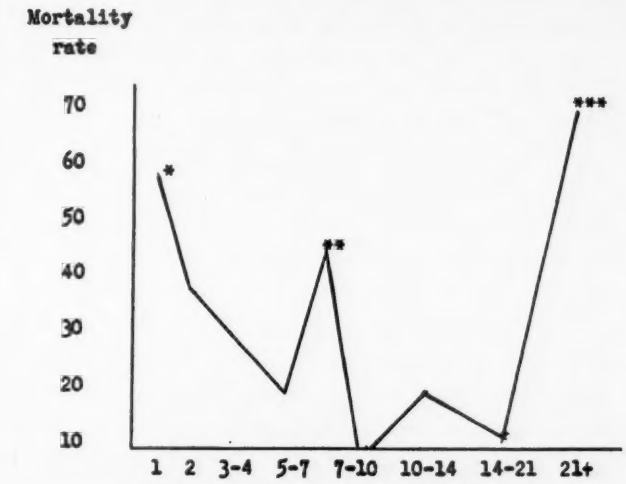
Perforated Cholecystitis (Pathology)	No. of Cases	Duration of Present Illness (before Admission)	Time in Hospital (before Operation)
With abscess (localized).	13	9.8 days (3–21 days*)	1.7 days (1 hr–6 days*)
Peritonitis secondary to rupture abscess.	10	16.1 days (2–50 days*)	3.3 days (2 hrs–13 days*)
Primary peritonitis (direct perforation).	30	6.9 days (2 hrs–26 days*)	1.2 days (2 hrs–6 days*)
Peritonitis complicated by severe pancreatitis.	7	2.2 days (12 hrs–5 days)	16 hours (1 hr–3 days*)

* Figures in parentheses represent the extremes in time element.

seen between three and seven days after the onset.

Table III cites the pertinent data on the immediate illness of patients operated on with perforations, and Table IV the data on their mortality.

GRAPH II Mortality rate in Perforated Cholecystitis on the basis of the pre-operative present illness.



* represents fulminating peritonitis with pancreatitis.
** represents perforation in delayed Gangrenous Cholecyst tis.
*** represents secondary peritonitis due to the rupture of a pericholecystic abscess.

TABLE IV
INFLUENCE OF DELAY IN SURGERY ON THE MORTALITY IN PERFORATED CHOLECYSTITIS (COMPARE WITH TABLE III)

Perforated Cholecystitis (Pathology)	No. of Deaths	Average Duration Present Illness	Average Time in Hospital	Per Cent Mortality
With abscess (localized)	0	.....	.....	0.0
Peritonitis secondary to rupture of abscess	4	27 days	6.5 days	40.0
Primary peritonitis (direct perforation)	10	7.5 days	1.4 days	33.3
Peritonitis complicated by severe pancreatitis	5	2.6 days	24 hours	71.4

The ten cases of peritonitis due to the secondary rupture of a previously localized abscess are of great interest. Delay in recognizing or draining a pericholecystic abscess was attended by a mortality of 40 per cent. These patients were admitted on an average of 16.1 days after the onset of their acute symptoms and subjected to a further average delay of 3.3 days before they came to operation. The cause of death is readily apparent; the four individuals who died were even longer delayed before they came to operation (total of thirty-four days). This would appear to be reliable evidence against the teaching of "watchful waiting," for had localization been suspected these patients would have been operated upon earlier. The "late peak" in mortality, shown in Graph II illustrates this danger.

The thirty cases with peritonitis from direct perforation of the gall-bladder had an average pre-hospitalization illness of 6.9 days and were observed 1.2 days. The mortality rate of 33.3 per cent is that generally reported for peritonitis in acute abdominal disease. Boyce et al.,[16] Liedberg,[20] Mentzer,[21] and others report a similar mortality. Glenn,[18] Heuer,[7] and Wolfson and Rothenberg[24] had a much lower mortality in acute perforated cholecystitis with earlier operation. This group of perforations occurred in neglected acute gangrenous cholecystitis.

The fourth group of perforated cholecystitis is that in which perforation

occurred associated with an acute suppurative or hemorrhagic pancreatitis. Little has been said of this highly fatal complication in the literature, but the cases in our series were definitely associated with a neglected chronic cholecystic disease and usually with common duct involvement. The early high incidence of perforation seen in Graph II is due to these cases. To determine whether or not an acute gall-bladder will perforate is as difficult as answering the same question in acute appendicitis. Touroff,[12] Heuer,[7] and Mitchell[13] have shown that there is little parallelism between the patient's clinical course and the pathologic progress of his acute infection. Forty-six of the perforations were observed in the hospital for twenty-four hours or more. Eleven of these did not appear to be progressive from a clinical standpoint, and yet they had a peritonitis. Four of these were localized and seven diffuse.

Cause of Death. There were nineteen deaths in this series of acute perforations. Table v is a summary of the causes of death and the time of death in relation to operation. Peritonitis was the cause of death directly or indirectly in two-thirds of the cases.

TABLE V
THE CAUSE AND TIME OF DEATH IN ACUTE PERFORATED CHOLECYSTITIS

Cause of Death	No. of Deaths	Time of Death (Days Post-operative)
Liver death	5	23 hours
Peritonitis	6	6.5 days
Peritonitis and pneumonia	3	7.0 days
Peritonitis and secondary hemorrhage	1	22 days
Pulmonary embolism	3	13 days
Uremia	1	16 days

CONCLUSIONS

1. Perforation of the gall-bladder during an acute attack is as frequent and as fatal from a statistical review as that in acute appendicitis.

2. Perforated cholecystitis in our cases did not localize as frequetly as has been reported in other series.

3. Perforation, other than that associated with a marked fulminating infection, was found when there was neglect in operating for chronic cholecystitis and delay in operating for acute cholecystitis.

4. The highest mortality in perforated cholecystitis was found in those patients with (1) pancreatitis, 71.4 per cent; (2) neglected acute gangrenous cholecystitis, 45 per cent; and (3) a perforation of a neglected pericholecystic abscess, 40 per cent.

5. A study of the clinical course and pathologic findings presented by those patients who recovered and those who died, reveals, as has been shown in other series, that "watchful waiting" is not a safe method because of the lack of parallelism between the clinical course of the disease and its pathologic progress.

6. We believe that when the high incidence of perforation and the dangers inherent in peritonitis due to acute cholecystitis are recognized, early surgery will become the general method of treatment.

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SURGERY OF THE COMMON DUCT*

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PATHOLOGY in and about the common bile duct is one of the causes for the mortality and morbidity encountered in biliary tract surgery. There has been a general reduction in the mortality and morbidity following surgery of the gall-bladder for simple non-complicated gall-bladder disease. This has been achieved through a better understanding as well as a consideration of the secondary changes associated with cholecystic disease. The factors of common duct pathology have, however, received less attention, and therefore, surgery on the ducts has not advanced as rapidly as has surgery on the gall-bladder. Recent interest and investigation in the field of common duct pathology promises better end results.

TABLE I

OPERATIONS FOR COMMON DUCT DISEASE

Operations	No. of Operations	No. of Deaths	Per Cent Mortality
Choledochostomy and cholecystectomy	327	40	12.2
Choledochostomy and cholecystostomy	25	9	36.1
Choledochostomy and cholecystectomy with a secondary operation	6	1	16.2
Choledochostomy secondary to a previous cholecystectomy	39	15	38.6
Cholecystogastrostomy (2 cholecystoduodenostomies)	52	15	28.8
Plastic on ducts	5	3	60.0
Total operations for duct pathology	454	83	18.2
Total biliary tract operations	3,986	309	7.7

This analysis of common duct surgery is an endeavor to determine some of the factors underlying the general high mortality and morbidity. The basis for this study is a series of 454 consecutive cases of common duct disease in which operation was done at the New York Post-Graduate Hospital between 1920 and July 1, 1937. Table I summarizes the operations performed, the number of deaths in each and the mortality rate.

Table II is a résumé of the same cases grouped in accordance to their postoperative diagnosis.

TABLE II

SURGERY ON THE BILE DUCTS POSTOPERATIVE DIAGNOSIS

	No. of Operations	No. of Deaths	Per Cent Mortality
Common duct stones			
And chronic cholecystitis	213	30	14.0
And acute cholecystitis	80	8	10.0
After cholecystectomy	33	12	36.4
Common duct disease without stones (dilated duct—cholangitis, stenosis, etc.)			
And chronic cholecystitis	11	1	9.0
And acute cholecystitis	11	3	27.0
After cholecystectomy	6	3	50.0
Chronic pancreatitis			
And chronic cholecystitis	26	1	3.8
And normal gall-bladder	6	1	16.2
Acute pancreatitis (suppurative and hemorrhagic)			
And chronic cholecystitis	8	2	25.0
And acute cholecystitis	7	5	71.4
Severe operative injury of the common duct (complete destruction of portion)	2	1	50.0
Carcinoma of the bile ducts	5	3	60.0
Carcinoma of the head of pancreas	46	14	30.4
Total operations for duct pathology	454	83	18.2

The common duct pathology found in the series falls into two groups, i.e., those conditions intrinsic to the duct, and those extrinsic to it. The diseases intrinsic to the duct are found associated with a pre-

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existing cholecystic disease in a majority of instances.

INTRINSIC COMMON DUCT DISEASE

Common Duct Stones. This was by far the most common cause for surgery on the common duct. Choledochostomy was performed at the time of cholecystectomy or cholecystostomy for chronic disease in 260 instances and stones were found in 82 per cent. In 3,306 operations for chronic cholecystitis, the incidence of common duct surgery was 7.7 per cent and of stones 6.3 per cent.

The invariable pathologic finding was an associated severe cholecystitis. This advanced pathology of the gall-bladder found in common duct disease lends authority to the assumption that intrinsic common duct disease is usually secondary to gall-bladder disease. That cholecystitis is a progressive disease which eventually involves the common duct has been noted repeatedly in our series. Walters[1] recently stated that "generally speaking, infection, stone formation, and obstruction of the common duct can be directly or indirectly related to inflammation which takes its origin in the gall-bladder." Lahey[2] is definitely of the belief that common duct infection, stone formation, and obstruction are due to neglected cholecystic disease. Lahey and Swinton[3] found stones in the gall-bladders of 98 per cent of their common duct stone cases.

It is probable that the more frequent open exploration of the common duct in our cases would have demonstrated a higher incidence of stones. Allen,[4] in a review of 1,228 cases, found that the common duct was explored in 32 per cent of them. Furthermore, stones were present in 13 per cent of all his cases. Cheever[5] also explored the common duct more frequently and in 426 personally operated cases, he did a choledochostomy on 37.9 per cent, finding stones in 44.3 per cent of those explored.

It is evident that if common duct surgery is to be avoided, the patient with chronic cholecystitis and cholelithiasis must come to surgery before the common duct has become involved. This means early diagnosis and early surgical intervention. In our series, the evidence of common duct stone and the mortality rate in operations on the common duct increased rapidly with the duration of the symptoms. (Table III.)

Table III
DURATION OF CHOLECYSTIC SYMPTOMS IN COMMON DUCT STONE

Duration of Symptoms	Total Cases	Common Duct No. Cases	Per Cent Common Duct Stone
Under 2 years	1,270	24	1.9
2–10 years	1,020	92	9.0
10–35 years	610	97	16.0

Delay in surgery is also a big factor in common duct stone associated with acute cholecystitis. In 574 operations for acute cholecystitis, eighty patients had common duct stones, an incidence of 14 per cent. In only three of these cases was there no history of previous cholecystic disease. Therefore 96 per cent had a definite chronic cholecystic disease history. Liedberg[6] found a similar incidence of common duct stones in 556 acute cases in which he operated. He believes that the common duct must be explored, if indicated in acute disease, in spite of the added risk, for often the common duct obstruction is the cause for the acute process.

Stones may be formed in the common duct after the removal of a pathologic gall-bladder. It is impossible to state how many of the thirty-three patients with the secondary common duct stone were of this type and in how many the stones had been overlooked at the previous operation. A number of the patients in this series were relatively symptom-free for three to ten years before they had recurrent symptoms. In six cases the common duct was literally filled with stones of the calcium bilirubinate type. Common duct stones were found in

80 per cent of all secondary common duct operations following cholecystectomy. This is in marked contrast to the report by Beye[7] in which only 6 or 9 per cent of sixty-six secondary operations showed common duct stones.

Intrinsic Disease without Stone. Stones were not found in thirteen cases of choledochostomy for chronic disease. These patients had a definite cholangitis, a stenosis of the common duct, or a marked spasm of the sphincter of Oddi. As in the stone cases, the gall-bladder showed severe pathology. Best and Hicken[8] believe dilatation of the common duct may result from persistent sphincter spasm. Walters[1] also is of this opinion.

A dilated common duct without evident pathology was found in eleven instances associated with an acute cholecystitis. In four cases the obstruction may have been due to a chronic pancreatitis. This was not confirmed. It is likely that cholangitis and stenosis were the causative factors.

In six cases of secondary choledochostomy stones were not found. Two of these had a severe cholangitis. The secondary operation followed shortly after a previous cholecystectomy. It is probable that the cholangitis was a residual infection of the preëxisting disease. Severe common duct stenosis was seen in three instances. This was due to operative injury. In two additional cases there were divided common ducts due to operative accidents.

Carcinoma of the Bile Ducts. Carcinoma was rare in this series. Only five proved cases came to operation. In three instances an excision of the cicatrix and duct anastomosis was attempted. One of the patients, at autopsy, showed a carcinoma of the ampulla of Vater. In the other two a high choledochostomy was done for temporary relief of icterus.

EXTRINSIC DISEASE OF THE COMMON DUCT

Carcinoma of the Head of the Pancreas. Carcinoma of the pancreas was the major cause for common duct surgery where the pathology lay extrinsic to the duct. Sixty-two cases were so diagnosed at operation, and only forty-six of these had short-circuiting operations. Cholecystostomy alone was done on the remainder. Cholecystogastrostomy was done in forty-four cases and cholecystoduodenostomy in two. The former was found to be highly satisfactory as a palliative measure. In the majority of the cases, operation was done when the disease was far advanced. The average icterus index was 114. Lahey,[9] in a recent publication, stated his belief that a sidetracking operation was of the greatest value. He, however, prefers a cholecystoenterostomy (jejunum) instead of the cholecystogastrostomy so generally used in our series. In none of the cases of this series was a resection of the head of the pancreas attempted. Best and Hicken[10] warn of the danger of a stenosis of the cystic duct in short-circuiting with the gall-bladder.

Chronic Pancreatitis. Chronic sclerosing pancreatitis is the second largest extrinsic cause of obstruction of the common duct. This condition was seen in thirty-six cases and contributes largely to those cases having a dilated common duct without other evidence of obstruction. These patients were, in the main, treated by choledochostomy. Again, a severe cholecystitis was usually associated with the pancreatitis. In four instances a fulminating acute cholecystitis was present with the chronic pancreatitis. Bergeret[11] found the incidence of chronic pancreatitis with common duct stone very high. He believes that the obstruction is the cause of pancreatitis.

Six cases of chronic pancreatitis were treated by means of a cholecystogastrostomy. In three a clinical diagnosis of carcinoma of the head of the pancreas had been made. In two this was disproved by a biopsy and in one at autopsy.

Acute Pancreatitis. Acute suppurative and hemorrhagic pancreatitis was present in fifteen cases, in eight associated with a chronic cholecystitis and in seven with an acute cholecystitis. The gall-bladder was

removed in each instance. Five of the acute cases were either gangrenous or perforated. Choledochostomy for acute pancreatitis is still a disputed procedure. Many operators practice drainage of the common duct in the presence of an associated cholecystitis. (Abell,[12] Dunlop and Hunt,[13] and Eliason and North.[14])

DIAGNOSIS

The surgeon has three opportunities for diagnosing common duct disease: a *preoperative diagnosis;* a *careful exploration* at the operation; and a careful study during the *postoperative period,* before removal of the drainage tube.

Preoperative Diagnosis. Jaundice, colic, chills, and fever are no longer considered pathognomonic of common duct stone or obstruction. A careful evaluation of the history and physical findings of the cases presented in this series shows that only 60 per cent had two or more of these symptoms. Jaundice was the most consistent finding. The reason for the frequent absence of colic was demonstrated by MacDonald.[15] He showed that typical colic cannot come primarily from the common duct.

Repeated duodenal drainages are of immeasurable value in the differential diagnosis of common duct disease. Persistent organisms, particularly colon bacilli, crystals, and pus cells, if found in the presence of a diseased gall-bladder, are strongly indicative of common duct stone. Stones rarely completely obstruct the common duct. In complete obstruction there is usually an extrinsic cause. Lahey's modification of Courvoisier's law is particularly apt in the diagnosis of carcinoma of the head of the pancreas. He states that "in the presence of jaundice which is persistent, painless, and progressive, together with consistently acholic stools when the gall-bladder is palpable, the obstruction is in practically all cases due to carcinoma of the head of the pancreas."

Diagnosis at the Time of Operation. Thorough exploration of the bile ducts and of the pancreas is an essential step in every operation on the biliary tract. Maingot,[16] MacDonald,[15] Walter,[1] Cattell,[17] Smyth and Mason,[18] and many others are advocating more frequent common duct exploration at the time of cholecystectomy. Nearly all agree that the common duct should be drained when once opened. Beall,[19] however, disagrees with this procedure. He believes that primary closure is the method of choice unless the obstruction cannot be removed. The use of cholangiography will materially reduce the hazard of overlooking common duct pathology.

The indications for exploring the common duct at operation are nicely summed up by Cutler and Zollinger.[20] They are (a) the suggestion of a stone on palpation, (b) a dilated or thickened duct, (c) a contracted gall-bladder, (d) a dilated cystic duct, (e) a thickening of the head of the pancreas, and (f) the presence of small stones in the gall-bladder or the cystic duct.

Postoperative Diagnosis. Every effort should be made to determine the function of the common duct before the drain is removed. Cholangiography will give the best index of the restoration of function of the common duct.

One of us (C. G. H.) has been employing the visualization of the common duct by the use of hippurin. The day before the test is to be made a preliminary injection of 20 to 30 c.c. of normal saline is injected through the T-tube. We have found that this amount will be taken with practically no discomfort. The quantity may be increased up to 40 c.c. with only some slight discomfort. The solution will run almost by the effect of gravity from the syringe into the common duct, and into the duodenum. On the following morning the patient is taken to the *x*-ray department and from 10 to 20 c.c. of sterile hippurin is injected slowly through the T-tube and an *x*-ray picture is taken immediately, a second picture four minutes and a third twelve minutes later. The first negative will show that both the right and left

hepatic ducts are well filled with the solution. There will be moderate distention of the common duct and, if the ampulla of Vater is unobstructed, the dye will be almost immediately present in the duodenum. In four minutes the dye will be in the duodenum and neighboring jejunal loops, and at the end of twelve minutes the third negative will show almost complete evacuation of the dye into the intestine.

The food test, as outlined by Carter,[21] is an excellent check on the function of the sphincter of Oddi. Careful microscopic and chemical examination of the bile from the common duct tube as well as the duodenal bile is of further help in assuring a normally functioning duct.

MORTALITY AND MORBIDITY

Of the 454 patients in this series, eighty-three died, a mortality of 18.2 per cent. Forty-six of the surviving patients had severe complications, potentially lethal. For the study of the mortality and morbidity, the series had been divided into four groups. They are (a) choledochostomy when combined with an operation for chronic cholecystitis; (b) choledochostomy when combined with an operation for acute cholecystitis; (c) choledochostomy secondary to a previous cholecystectomy; and (d) cholecystogastrostomy for obstruction of the lower common duct.

Choledochostomy Associated with Chronic Cholecystitis. Surgical drainage of the common duct was performed 254 times in the presence of chronic cholecystitis. The mortality was 13 per cent, a rate much higher than that reported by Eliason and Erb,[22] Allen,[4] Lahey,[2] and Cheever.[5] Table IV is a summary of the cause of death and the postoperative morbidity in this group. The common duct stone cases had generally far advanced cholecystic disease. These included twelve cases in which there was a chronically perforated gall-bladder. Six of these patients died. The mortality was primarily influenced by the age of the patient, the duration of the illness, and the severity of the infection.

Pneumonia leads as the cause of death. Pneumonia and cardiac failure were found predominantly in patients over 60 years of age. Peritonitis, the cause of death in seven cases, resulted from perforation of the gall-bladder in three instances and from acute pancreatitis in three. Those diagnosed as "liver death" died of the typical syndrome described by Heyd[23] in 1924.

Delay and deferred surgery in chronic cholecystitis and cholelithiasis is the primary cause for the high mortality and morbidity reported. The high incidence of pneumonia and cardiac failure in the aged, the number of perforations as well as the incidence of pancreatitis, and the large number of infections, all point to delay in operating.

Choledochostomy Associated with Acute Cholecystitis. Of the ninety-six patients in this series sixteen died, a mortality of 16.4 per cent. The incidence of common duct disease coexistent with acute cholecystitis was 17 per cent. Table V is a summary of the cause of death and the morbidity in this group. Most of the patients requiring common duct surgery in acute disease had a history of repeated attacks of colic or inflammation.

The major cause of death in this group was peritonitis, with pneumonia second in frequency. All but one of the deaths due

TABLE IV
CHOLEDOCHOSTOMY AND CHRONIC CHOLECYSTITIS

Cause of Death		Major Complications	
Pneumonia	8	Wound infection	5
Peritonitis	7	Postoperative hemorrhage	5
Liver death	7	Pneumonia	3
Cardiac failure	5	Dehiscence	3
Postoperative hemorrhage	2	Thrombophlebitis	3
Uremia	2	Intraperitoneal infection	3
Pulmonary embolism	2		
Coronary occlusion	1		

TABLE V
CHOLEDOCHOSTOMY AND ACUTE CHOLECYSTITIS

Cause of Death		Severe Complications	
Peritonitis	7	Wound infection	4
Pneumonia	4	Pneumonia	2
Liver death	2	Peritonitis	3
Postoperative hemorrhage	1	Persistent fistula	1
Cardiac failure	1		
Coronary occlusion	1		

to peritonitis occurred in patients who had gangrenous or perforated gall-bladders complicating their common duct disease. Pneumonia was again seen in the aged.

The severe complications were due in the main to infection. In those patients with common duct disease and acute cholecystitis, where the process had not progressed to empyema, gangrene, or perforation, the mortality was 7.2 per cent. Delay in operating in chronic cholecystitis increased the incidence of acute cholecystitis and the incidence of common duct involvement; and delay in operating during an acute attack increased the severity of the acute pathology. These factors probably account for the mortality and morbidity in this group of patients.

Secondary Choledochostomy. Of the thirty-nine patients who underwent secondary choledochostomy, fifteen died, a mortality of 38.6 per cent. Table VI is a summary of the causes of death and the severe complications encountered in this group of patients. The mortality is identical with that experienced in most clinics. The high mortality rate in this group emphasizes the importance of discovering and treating common duct disease at the time of the primary operation.

Cholecystogastrostomy. Fifteen of the fifty-two patients in whom a cholecystogastrostomy or duodenostomy was done, died, a mortality of 28.8 per cent. Forty-six of these were for carcinoma of the head of the pancreas. The mortality in our series is that generally reported. Bernhard[25] reports a mortality of 29.8 per cent, Eliason and Erb[22] 38.4 per cent, Mayo-Robson[26] 53.3 per cent, and Kehr[27] 75 per cent. Best and Hicken[10] found a mortality of 41.1 per cent in 221 cases reported in the literature.

The average age of these patients (entire series) was 60 years. Table VII shows that pneumonia was the major cause of death, which was to be anticipated in the aged and debilitated patients. Only three deaths can be attributed to operative difficulty, two because of operative shock and one peritonitis. The death from hemorrhage occurred in reoperation. No bleeding point was determined, the hemorrhage being general. The icteric index in this patient was 240 preoperatively.

Table VI

SECONDARY CHOLEDOCHOSTOMY

Cause of Death		Severe Complications	
Peritonitis	5	Wound infections	2
Liver death	3	Pneumonia	1
Pneumonia	3	Peritonitis	1
Operative shock	2		
Cardiac failure	2		

Table VII

CHOLECYSTOGASTROSTOMY

Cause of Death		Severe Complications	
Pneumonia	9	Hemorrhage	4
Liver death	2	Dehiscence	2
Operative shock	2		
Peritonitis	1		
Postoperative hemorrhage	1		

CONCLUSIONS

1. Delay in operating on patients with chronic cholecystitis and cholelithiasis is the major cause for common duct disease.
2. Repeated acute attacks of cholecystitis predispose to acute common duct pathology.
3. The mortality and morbidity in surgery of the common duct are directly proportional to the duration of the symptoms, the age of the patient, and the severity of the associated cholecystic disease.
4. The common duct should be explored whenever any probable indication of disease is present.
5. Secondary choledochostomy carries a very high mortality. This high mortality justifies more frequent primary exploration.
6. The mortality of cholecystogastrostomy is due to the general condition of the patient, especially jaundice and the factor of malignancy. Earlier diagnosis and surgery can reduce this mortality.

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PANCREATITIS AND BILIARY TRACT DISEASE*

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A DEFINITE relationship between biliary tract disease and pancreatitis has long been recognized. Korte's[1] treatise in 1898 called attention to acute necrotic pancreatitis as associated with biliary tract disease. Fitz,[2] in 1889, first thought pancreatitis due to a "gastroduodenitis," but later (1899) came to believe it due to a common duct lesion. Opie,[3] in 1901, showed acute necrotic pancreatitis to be due to a regurgitation of bile into the pancreatic ducts. His conclusions have been ably seconded by Archibald[4] and many others. This view has been generally held because of the frequent association of the two lesions (20 per cent to 50 per cent—Jones[5]). More recently the lymphatic origin of all types of pancreatitis has been indicated both experimentally and clinically. Graham and Peterman[6] have been outstanding advocates of this theory.

Regardless of the actual etiology of chronic and acute pancreatitis, biliary tract disease pathology is frequently found coëxistent with a pancreatitis and therefore its treatment enters into the surgical considerations to be weighed when pancreatitis is operated upon. Cullen and Friedenwald,[7] Takats and MacKenzie,[8] Babcock,[9] Dragstedt, Haymond, and Ellis,[10] Rich and Duff,[11] Elman,[12] Walters,[13] and Bergeret,[14] are but a few who have, in the recent literature discussed this relationship and have indicated that the surgical treatment must include drainage of the biliary tract as well as a direct attack on the pancreas.

We are presenting forty-seven cases of proved biliary tract disease with associated pancreatitis operated upon at the New York Post-Graduate Hospital from 1920 to July, 1937. These cases of pancreatitis were found as a part of a study of 3,986 operations for biliary tract disease. An additional six cases had common duct surgery done for chronic pancreatitis, but no evidence of biliary tract disease was noted.

Table 1 indicates the postoperative diagnosis in cases with pancreatitis with associated biliary tract disease.

CHRONIC PANCREATITIS

Chronic pancreatitis was diagnosed in the presence of chronic cholecystic disease in thirty cases. Two-thirds of these had common duct stones. The operator found the common duct stone resting at, or impacted in, the ampulla of Vater in fifteen of these cases (75 per cent). This observation lends clinical evidence to the theory that regurgitation of bile into the pancreatic ducts may well play a rôle in the cause of the pancreatitis encountered in these cases. All but one of the patients with pancreatitis and biliary tract disease had a long standing history of cholecystic disease (average eleven years). The gall-bladder pathology was uniformly severe. Ulceration, fibrosis, hydrops, and chronic empyema were common pathologic findings. Nearly 50 per cent of these cases gave clinical histories and pathologic findings suggestive of previous acute cholecystitis.

The six cases with a normal gall-bladder are of considerable interest, for here the common duct obstruction was due to a pancreatitis without an existing common duct disease. These patients were treated by means of a cholecystogastrostomy. In three a diagnosis of carcinoma of the head of the pancreas was made at the time of operation. In two the diagnosis of carci-

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noma was disproved by biopsy and in the third by autopsy.

ACUTE PANCREATITIS

Acute pancreatitis was found associated with definite cholecystitis in seventeen instances. In fifteen the clinical pathologic diagnosis was that of an acute hemorrhagic or suppurative pancreatitis. Only two cases of acute interstitial pancreatitis were diagnosed. Chronic cholecystitis was seen in ten instances, associated with a common duct stone in eight instances. The past history of these cases was generally similar. All had a past history of chronic cholecystic symptoms of long duration with occasional jaundice (average thirteen years). The acute episode was sudden, dramatic, severe. They came to operation an average of fifty-two hours after the onset of acute symptoms.

The seven cases with acute cholecystitis and acute pancreatitis were particularly severe. Five had a gangrenous perforated gall-bladder complicating the fulminating pancreatitis. In each instance the patient had a history of chronic cholecystic symptoms. Stones were present in the gall-bladder or free in the peritoneal cavity in each case and, in addition, common duct stones were found in five. The patients were operated upon on an average of 2.8 days after the onset of their acute attack and after having been observed in the hospital an average of sixteen hours.

TABLE I
POSTOPERATIVE DIAGNOSIS OF FIFTY-THREE CASES OPERATED UPON FOR PANCREATITIS WITH BILIARY TRACT DISEASE

	No. of Cases	Deaths	Per Cent Mortality
Chronic pancreatitis			
With chronic cholecystitis...	10	1	10
With chronic cholecystitis and common duct stone...	20	1	5
Normal biliary tract.....	6	1	16.6
Total chronic pancreatitis.....	36	3	8.3
Acute pancreatitis			
With chronic cholecystitis...	2	2	100
With chronic cholecystitis and common duct stone...	8	2	25
With acute cholecystitis.....	7	5	71
Total acute pancreatitis.....	17	9	53

OPERATIVE PROCEDURE

Table II is a résumé of the operative procedures undertaken in the cases here reported.

Drainage of the common duct is essential in the treatment of acute and chronic pancreatitis if any evidence of obstruction is present. In the majority of cases this is best accomplished by means of a choledochostomy. (Table II.) Cholecystostomy was resorted to only six times, four in chronic and two in acute cases, with a very unfavorable mortality. Best and Hicken[17] warn of the danger of using the gall-bladder for draining the common duct unless the patency of the cystic duct has been established. Cholecystogastrostomy was successfully performed in five out of six cases where the obstruction was so severe as to make a choledochostomy of questionable efficacy. Care must be taken to differentiate a chronic pancreatitis from a carcinoma of

TABLE II
RÉSUMÉ OF THE VARIOUS OPERATIVE PROCEDURES UNDERTAKEN FOR THE RELIEF OF PANCREATITIS

	No. of Cases	Deaths	Per Cent Mortality
For chronic pancreatitis			
Cholecystostomy.....	4	1	25
Cholecystectomy and choledochostomy.....	26	1	3.8
Cholecystogastrostomy.....	6	1	16.6
Total chronic pancreatitis.....	36	3	8.3
For acute pancreatitis			
Cholecystostomy.....	2	2	100
Cholecystectomy and choledochostomy.....	15	7	47
Total acute pancreatitis.....	17	9	53.3

the head of the pancreas so that the more radical short-circuiting operation may be avoided.

Acute pancreatitis presents a therapeutic dilemma that necessitates the greatest care and judgment if the patient is to survive. The treatment in our series was uniform in spite of the fact that eight surgeons operated on these cases. In each instance the capsule of the pancreas was incised and multiple drains inserted. Then cholecystectomy and dochostomy were performed except in the two instances of cholecystostomy.

The high incidence of common duct stone in both chronic and acute pancreatitis, the severe gall-bladder pathology generally found, and the long duration of the chronic history, all suggest delay in the treatment of the preëxisting chronic cholecystitis to be the fundamental cause for the pancreatitis encountered in this series. Takats and MacKenzie,[8] Cullen and Friedenwald,[7] Elman,[12] Pratt,[18] and Heyd[19] are among those who believe that the majority of cases of acute hemorrhagic and suppurative pancreatitis result from neglected cholecystic disease. Early diagnosis and surgical treatment should reduce the incidence of acute and chronic pancreatitis as well as the high mortality found in it.

MORTALITY

The mortality rate in chronic pancreatitis and chronic cholecystitis was 8.3 per cent. This was only slightly higher than that found in the general series of chronic cholecystic disease (6.4 per cent). The mortality rate for choledochostomy in the presence of a chronic pancreatitis was only 3.8 per cent, a markedly lower rate than that seen in the 238 choledochostomies done for all chronic causes (11.34 per cent).

In acute pancreatitis, however, the mortality rate is exceedingly high. In our series there was a general mortality rate of 53.3 per cent. Those cases with chronic cholestitis had a mortality rate of 40 per cent and those with acute cholecystitis had one of 71 per cent. This high mortality is that generally reported in the literature. From Table II it can be seen that where the common duct has been drained, the mortality rate is more favorable than in those cases with indirect drainage. This may be a factor in reducing the mortality in all types of pancreatitis.

Peritonitis is the predominant cause for this high mortality. Eleven of the twelve deaths seen in our series were due to peritonitis. Six of these were autopsied, thus proving this diagnosis. Only one death was attributable to liver death (hepatic insufficiency). As no autopsy was performed it may well have been due to a fulminating intraperitoneal infection.

SUMMARY

Fifty-three cases of chronic and acute pancreatitis on whom biliary tract surgery was performed have been discussed. Biliary tract disease was present in all but six of these. Common duct stone was found in 66 per cent of those with biliary tract disease and chronic pancreatitis and in 78 per cent of those with acute pancreatitis. The surgical treatment, in the vast majority of cases, involved a drainage of the common duct as well as a direct attack on the pancreas. The mortality rate in chronic pancreatitis is that inherent in surgery on the biliary tract, and the mortality rate in acute pancreatitis that found in severe peritonitis. Peritonitis was the predominant cause of death in all cases.

The prophylaxis of both chronic and acute pancreatitis lies in the early surgical treatment of the preëxisting biliary tract infection.

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